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NEWS 34	Apr 21	Indexing from 1947 to 1956 being added to records in CA/CAPLUS
NEWS 35	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX

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FILE 'HOME' ENTERED AT 15:16:06 ON 21 APR 2003

=> file medline embase biosis scisearch caplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 0.21 0.21

FILE 'MEDLINE' ENTERED AT 15:16:22 ON 21 APR 2003

FILE 'EMBASE' ENTERED AT 15:16:22 ON 21 APR 2003

FILE 'BIOSIS' ENTERED AT 15:16:22 ON 21 APR 2003

FILE 'SCISEARCH' ENTERED AT 15:16:22 ON 21 APR 2003

FILE 'CAPLUS' ENTERED AT 15:16:22 ON 21 APR 2003

=> s schizophrenia diagnosis
L1 422 SCHIZOPHRENIA DIAGNOSIS

=> s l1 and DTH
L2 1 L1 AND DTH

=> d l2 cbib abs

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
1999:390463 Document No. 131:16115 Skin test for schizophrenia. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9930163 A1 19990617, 21 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-IL592 19981207. PRIORITY: IL 1997-122490 19971207.
AB A diagnostic method for assaying schizophrenia in a subject is provided wherein a prepn. comprising platelet derived proteins or fractions thereof

having a pI above about 6.5 is injected into a subject and the occurrence of delayed type hypersensitivity (**DTH**) reaction at the site of the injection is detd. A pos. **DTH** reaction indicates that the tested subject has a high likelihood of being schizophrenic. The protein prepn. used in the diagnostic method is also provided as well as a method for its prepn. and a kit for use in the diagnosis of schizophrenia using the above method.

=> s l1 and platelet
L3 4 L1 AND PLATELET

=> dup remove l3
PROCESSING COMPLETED FOR L3
L4 4 DUP REMOVE L3 (0 DUPLICATES REMOVED)

=> d l4 1-4 cbib abs

L4 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2003 ACS
2002:736274 Document No. 137:259655 Novel peptides for the diagnosis of schizophrenia. Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 2002074793 A2 20020926, 27 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-IL233 20020321. PRIORITY: IL 2001-142159 20010321; US 2001-PV278659 20010321.
AB Short peptides are provided, which bind to a body fluid sample obtained from a schizophrenic patient at a substantively higher level than to a body fluid sample obtained from a non-schizophrenic individual. The peptides are no more than 10 amino acids long and comprise a continuous sequence of at least 5 amino acids which consists of at least one pos. charged amino acid at one of its ends. The provided peptides, which are the putative binding sites of autoantibodies found in high levels in schizophrenic individuals, are thus useful in diagnosis of schizophrenia. Biotin-labeled peptide LVVGLCK was coated onto streptavidin-coated tubes and used to test plasma samples of schizophrenic patients and control non-schizophrenic patients in an enzyme immunoassay.

L4 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2003 ACS
1999:390463 Document No. 131:16115 Skin test for schizophrenia. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9930163 A1 19990617, 21 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-IL592 19981207. PRIORITY: IL 1997-122490 19971207.
AB A diagnostic method for assaying schizophrenia in a subject is provided wherein a prepn. comprising **platelet** derived proteins or fractions thereof having a pI above about 6.5 is injected into a subject and the occurrence of delayed type hypersensitivity (DTH) reaction at the site of the injection is detd. A pos. DTH reaction indicates that the tested subject has a high likelihood of being schizophrenic. The protein

prepn. used in the diagnostic method is also provided as well as a method for its prepn. and a kit for use in the diagnosis of schizophrenia using the above method.

L4 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2003 ACS

1993:18858 Document No. 118:18858 Method of diagnosing or categorizing disorders from biochemical profiles. Matson, Wayne R. (ESA, Inc., USA). PCT Int. Appl. WO 9213273 A1 19920806, 42 pp. DESIGNATED STATES: W: CA, JP, RU; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1992-US375 19920116. PRIORITY: US 1991-643541 19910118; US 1991-649676 19910201.

AB A method for diagnosing disorders in living organisms is disclosed, in which fluid samples from normal and afflicted (abnormal) individuals are analyzed to generate patterns representative of mol. constituents of said samples. A data base of frequency distribution patterns of constituents of samples from organisms having known categories of disorders and controls is created, and the unknown sample anal. is compared for conformity to the frequency distribution patterns. The invention has particular applicability to diagnosing diseases, e.g. Alzheimer's disease, Parkinson's disease, Huntington's disease, schizophrenia, progressive supranuclear palsy, amyotrophic lateral sclerosis, and senile dementia. The invention also may be advantageously employed to diagnose diseases such as tumors, carcinomas, cardiovascular abnormalities, and other disorders, or for selection of the therapy based on categories of known vs. unsuccessful outcomes. Moreover, both treatment protocols and new pharmaceuticals may be evaluated. Cerebrospinal fluid samples from patients with Alzheimer's disease, Parkinson's disease, schizophrenia, Huntington's disease, and supranuclear palsy and from neurol. normal controls were analyzed by chromatog. and a 16-sensor electrochem. cell for 38 known components (e.g. adenine, cysteine, tyramine, uric acid, etc.) and for 18 well-defined unknown peaks. Linear and stepwise regression anal. were used in preliminary evaluation of the data and then cluster anal. procedures were performed. The biochem. response of controls or normal individuals was more chaotic then that of disordered individuals. Frequency distribution graphs of Alzheimer's disease and controls were prepd. as well as a plot showing scoring of Alzheimer's vs. control.

L4 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1981:271523 Document No.: BA72:56507. A BIOCHEMICAL STUDY OF TARDIVE DYS KINESIA IN YOUNG MALE PATIENTS. JESTE D V; DELISI L E; ZALCMAN S; WISE C D; PHELPS B H; ROSENBLATT J E; POTKIN S G; BRIDGE T P; WYATT R J. ADULT PSYCHIATRY BRANCH, DIV. OF SPECIAL MENTAL HEALTH RES., INTRAMURAL RES. PROGRAM, NATIONAL INST. OF MENTAL HEALTH, SAINT ELIZABETHS HOSP., WASHINGTON, DC 20032.. PSYCHIATRY RES, (1981) 4 (3), 327-332. CODEN: PSRSDR. ISSN: 0165-1781. Language: English.

AB Based on specific criteria, tardive dyskinesia was diagnosed in 6 of 29 young schizophrenic male inpatients. Several biochemical parameters in these 6 dyskinesia patients were compared with those in 6 matched controls. The patients with dyskinesia had significantly lower **platelet** monoamine oxidase activity and significantly higher plasma dopamine-.beta.-hydroxylase activity as compared with the controls, thus confirming the previous findings in a population of elderly female inpatients. The dyskinetic and nondyskinetic groups did not differ from each other in mean whole blood serotonin concentration and mean serum neuroleptic (thioridazine, mesoridazine haloperidol, fluphenazine, chlorpromazine, thiothixene) concentration as measured with a radioreceptor binding assay.

=> d his

(FILE 'HOME' ENTERED AT 15:16:06 ON 21 APR 2003)

FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 15:16:22 ON 21 APR 2003

L1 422 S SCHIZOPHRENIA DIAGNOSIS
L2 1 S L1 AND DTH
L3 4 S L1 AND PLATELET
L4 4 DUP REMOVE L3 (0 DUPLICATES REMOVED)

=> s l1 and pI
L5 2 L1 AND PI

=> dup remove l5
PROCESSING COMPLETED FOR L5
L6 2 DUP REMOVE L5 (0 DUPLICATES REMOVED)

=> d l6 1-2 cbib abs

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
1999:390463 Document No. 131:16115 Skin test for schizophrenia. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9930163 A1 19990617, 21 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-IL592 19981207. PRIORITY: IL 1997-122490 19971207.

AB A diagnostic method for assaying schizophrenia in a subject is provided wherein a prepn. comprising platelet derived proteins or fractions thereof having a **pI** above about 6.5 is injected into a subject and the occurrence of delayed type hypersensitivity (DTH) reaction at the site of the injection is detd. A pos. DTH reaction indicates that the tested subject has a high likelihood of being schizophrenic. The protein prepn. used in the diagnostic method is also provided as well as a method for its prepn. and a kit for use in the diagnosis of schizophrenia using the above method.

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
2000:142947 Document No. 133:116929 Frontal lobe in vivo 31P-MRS reveals gender differences in healthy controls, not in schizophrenics. Riehemann, Stefan; Volz, Hans-Peter; Wenda, Berit; Hubner, Gabriele; Rossger, Grit; Rzanny, Reinhard; Sauer, Heinrich (Department of Psychiatry, University of Jena, Jena, D-07740, Germany). NMR in Biomedicine, 12(8), 483-489 (English) 1999. CODEN: NMRBEF. ISSN: 0952-3480. Publisher: John Wiley & Sons Ltd..

AB Phosphorus-31 magnetic resonance spectroscopy (31P-MRS) has gained much interest in schizophrenia research in recent years since it allows the non-invasive measurement of high-energy phosphates and phospholipids in vivo. However, until now only differences in metabolite concns. between certain brain areas of schizophrenic patients and healthy controls have been examd. We investigated the influence of gender on the concns. of different phosphorus compds. For this purpose, well-defined vols. in the frontal lobe of 32 healthy controls and 51 schizophrenic in-patients were examd. with an image selected in vivo spectroscopy (ISIS) sequence on a whole-body scanner at 1.5 T. Healthy females exhibited increased values of inorg. phosphate (**Pi**) and decreased values of phosphocreatine (PCr) in comparison to their male counterparts. In schizophrenic patients such gender differences were not present. Thus, the results can be interpreted in the sense that frontal energy demanding processes are enhanced in female compared to male healthy volunteers; schizophrenia

seems to reduce these gender differences.

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=> dup remove l1
PROCESSING COMPLETED FOR L1
L7          326 DUP REMOVE L1 (96 DUPLICATES REMOVED)

=> s l7 and platelet protein
L8            0 L7 AND PLATELET PROTEIN

=> s demented patient
L9         5690 DEMENTED PATIENT

=> s l9 and "alzheimer's type dementia"
L10           0 L9 AND "ALZHEIMER'S TYPE DEMENTIA"

=> s dementia
L11      143369 DEMENTIA

=> s l11 and alzheimer
L12       60675 L11 AND ALZHEIMER

=> s l12 and platelet
L13         463 L12 AND PLATELET

=> s l13 and pI
L14           0 L13 AND PI

=> s l13 and "pI"
L15           0 L13 AND "PI"

=> dup remove l13
PROCESSING COMPLETED FOR L13
L16         251 DUP REMOVE L13 (212 DUPLICATES REMOVED)

=> s l16 and DHT
L17           0 L16 AND DHT

=> s l16 and shinitzky
L18           0 L16 AND SHINITZKY

=> s l16 and diagnosis
L19          47 L16 AND DIAGNOSIS

=> dup remove l19
PROCESSING COMPLETED FOR L19
L20           47 DUP REMOVE L19 (0 DUPLICATES REMOVED)

=> d l20 1-47 cbib abs

L20  ANSWER 1 OF 47  CAPLUS  COPYRIGHT 2003 ACS
2002:927573   Document No. 138:12497  Purification of astrocyte precursor cells
     and other lineage-specific cells from mammalian CNS on the basis of
     differential marker expression, their use in tissue replacement therapy,
     and other therapeutic and diagnostic applications.  Tailoi, Chan-Ling
     (University of Sydney, Australia).  PCT Int. Appl. WO 2002097069 A1
     20021205, 71 pp.  DESIGNATED STATES: W:  AE, AG, AL, AM, AT, AU, AZ, BA,
     BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE,
     ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR,
     KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ,
     OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
     UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM;
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RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-AU704 20020531. PRIORITY: US 2001-PV295455 20010601.

AB The invention relates to a method for developing a population of substantially lineage-specific cells and their use in tissue replacement therapy, tissue augmentation therapy, diagnostic applications, for the identification of growth factors and other autocrine factors. Specifically, substantially homogeneous populations of human and other mammalian cells of the astrocyte lineage are provided and selected on the basis of differential marker expression. The present invention identifies astrocyte cell markers which are capable of distinguishing between developmental stages. From multipotent stem cells, lineage-specific astrocyte precursor cells (APCs) are formed expressing Pax2 and vimentin but not either glial fibrillary acid protein (GFAP) or S-100. The next developmental stage is the formation of immature perinatal astrocytes (IPAs) which express all four of the above markers. Mature perinatal astrocytes (MPAs) lose the ability to express vimentin and then adult astrocytes further lose Pax2 expression as a function of physiol. aging. The ability to selectively enrich cultures of cells for APCs or IPAs permits their use in tissue replacement and augmentation therapy. Importantly, APCs have been identified in accordance with the present invention in adult brain as well as the retina and, hence, this aspect represents a source of APCs for autologous therapy as well as for heterologous therapy. Furthermore, homogeneous populations of APCs or IPAs can be used to isolate particular growth or autocrine factors for use in conjunctive therapy to tissue replacement and augmentation therapy or to induce repair or regeneration of endogenous tissue. The markers further permit mixed populations of astrocytes in various stages of development to be identified and this has diagnostic and therapeutic applications.

L20 ANSWER 2 OF 47 CAPLUS COPYRIGHT 2003 ACS

2002:906554 Document No. 138:1044 G protein-coupled receptor (GPCR) microarrays for determination of GPCR gene expression profiles and uses in drug and toxin screening and diagnostics. Thirstrup, Kenneth; Madsen, Lars Siim; Jensen, Jens Bitsch; Hummel, Rene; Jensen, Bo Skaaning (Azign Bioscience A/s, Den.). PCT Int. Appl. WO 2002095065 A2 20021128, 43 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK337 20020521. PRIORITY: DK 2001-802 20010518.

AB The invention provides G protein-coupled receptor (GPCR) arrays, kits comprising GPCR arrays and methods to produce such GPCR arrays. GPCR arrays are useful in the detn. of GPCR expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved in the response of the GPCR expression. The invention relates to an GPCR array comprising a multiplicity of individual GPCR polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual GPCR polynucleotide spot comprises an GPCR polynucleotide compn. comprising a non-conserved region of an GPCR polynucleotide family member, the spots representing at least two different regions of an GPCR polynucleotide member of a family. The invention also relates to a set of primers specific for nonconserved regions of GPCR polynucleotide family members, wherein the set of primers are used in the method for the prodn. of an array according to the invention. In still a further aspect, the invention relates to a

diagnostic method to det. the differences of GPCR expression profiles between two different biol. materials.

L20 ANSWER 3 OF 47 CAPLUS COPYRIGHT 2003 ACS
2002:906553 Document No. 138:1043 Transporter microarrays for the determination of transporter gene expression profiles and uses in drug and toxin screening and diagnostics. Jensen, Jens Bitsch; Madsen, Lars Siim; Gether, Ulrik; Jensen, Bo Skaaning (Azign Bioscience A/S, Den.). PCT Int. Appl. WO 2002095064 A1 20021128, 41 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK336 20020521. PRIORITY: DK 2001-803 20010518.

AB The object of the invention is to provide transporter arrays, kits comprising transporter arrays and methods to produce such transporter arrays. Transporter arrays are useful in the detn. of transporter expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the transporter expression. The invention relates to an transporter array comprising a multiplicity of individual transporter polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual transporter polynucleotide spot comprises an transporter polynucleotide compn. comprising a non-conserved region of an transporter polynucleotide family member, the spots representing at least two different regions of a transporter polynucleotide. A set of primers specific for nonconserved regions of transporter polynucleotide family members are provided, wherein the set of primers are used in the method for the prodn. of an array according to the invention. A diagnostic method detecting the differences of transporter expression profiles between two different biol. materials is also provided.

L20 ANSWER 4 OF 47 CAPLUS COPYRIGHT 2003 ACS
2002:832908 Document No. 137:347474 Ion channel microarrays for the determination of ion channel gene expression profiles and uses in drug and toxin screening and diagnostics. Jensen, Bo Skaaning; Madsen, Lars Siim; Jensen, Jens Bitsch; Kjaer, Katrine (Neurosearch A/S, Den.). PCT Int. Appl. WO 2002086050 A2 20021031, 53 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK253 20020418. PRIORITY: DK 2001-635 20010420.

AB The invention provides completely novel and improved ion channel arrays, kits comprising ion channel arrays and methods to produce such ion channel arrays. Ion channel arrays are useful in the detn. of ion channel expression profiles in a certain biol. material, several biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the ion channel expression. In a first aspect the invention relates to an ion channel array comprising a multiplicity of individual ion channel polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual ion channel polynucleotide spot comprises an ion channel polynucleotide compn. comprising a non-conserved region of an ion

channel polynucleotide family member, the spots representing at least two different regions of an ion channel polynucleotide member of a family. In a further aspect, the invention relates to a set of primers specific for nonconserved regions of ion channel polynucleotide family members, wherein the set of primers are used in the method for the prodn. of an array according to the invention. In still a further aspect, the invention relates to a diagnostic method to det. the differences of ion channel expression profiles between two different biol. materials; said method comprises obtaining a first ion channel expression profile of a first biol. material according to the method of the present invention, obtaining a second ion channel expression profile of a second biol. material according to the method of the present invention, comparing the first and second ion channel expression profiles, and identifying any difference in the ion channel expression profile.

L20 ANSWER 5 OF 47 MEDLINE

2003016227 Document Number: 22410662. PubMed ID: 12522675. Early stages of probable **Alzheimer** disease are associated with changes in **platelet** amyloid precursor protein forms. Borroni B; Colciaghi F; Corsini P; Akkawi N; Rozzini L; Del Zotto E; Talarico G; Cattabeni F; Lenzi G L; Di Luca M; Padovani A. (Clinica Neurologica, Dipartimento di Scienze Mediche e Chirurgiche, Universita degli Studi di Brescia, Piazza Ospedale 1, Italy.) NEUROLOGICAL SCIENCES, (2002 Dec) 23 (5) 207-10. Journal code: 100959175. ISSN: 1590-1874. Pub. country: Italy. Language: English.

AB Previous findings demonstrated an altered pattern of amyloid precursor protein (APP) forms in **platelets** of **Alzheimer** disease (AD) patients, compared both with healthy control subjects or patients with non-**Alzheimer**-type **dementia**. The present study aims to evaluate whether **platelet** APP form ratio (APPr) is altered in patients with early stage AD. We selected 40 patients with early stage AD and 40 age-matched healthy controls. Compared with controls (mean+/-SD=0.91+/-0.3), mean APPr was decreased in AD (mean+/-SD=0.46+/-0.26, p<0.0001). Sixteen very mild AD patients (clinical **dementia** rating=0.5), identified among the AD group, showed a significant decrease of APPr values (mean+/-SD=0.50+/-0.3, p<0.0001). These findings indicate that alteration of APP processing in **platelets** is an early event and suggest that this assay might be of diagnostic value in differentiating mild AD from normal ageing.

L20 ANSWER 6 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)

2002:752754 The Genuine Article (R) Number: 592JQ. Peripheral blood abnormalities in **Alzheimer** disease: Evidence for early endothelial dysfunction. Borroni B; Volpi R; Martini G; Del Bono R; Archetti S; Colciaghi F; Akkawi N M; Di Luca M; Romanelli G; Caimi L; Padovani A (Reprint). Univ Brescia, Spedali Civili Brescia, Dept Neurol, Neurol Clin 2, Brescia, Italy (Reprint); Univ Brescia, Dept Biochem, Brescia, Italy; Univ Brescia, Dept Med, Brescia, Italy; Dept Biotechnol, Lab 3, Brescia, Italy; Univ Milan, Inst Pharmacol Sci, Milan, Italy. ALZHEIMER DISEASE & ASSOCIATED DISORDERS (JUL-SEP 2002) Vol. 16, No. 3, pp. 150-155. Publisher: LIPPINCOTT WILLIAMS & WILKINS. 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA. ISSN: 0893-0341. Pub. country: Italy. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Clinical and epidemiologic studies demonstrate that vascular risk factors may be involved in **Alzheimer** disease (AD). To evaluate whether vascular abnormalities are an early feature of AD, several parameters of coagulation and fibrinolysis were assessed. Thirty patients with mild AD and 30 age-matched control subjects entered the study. All subjects performed a standardized clinical and laboratory protocol. Persons with vascular risk factors and systemic diseases were excluded. AD patients present significant increased levels of thrombomodulin (p <

0.0001) and sE-selectin ($p < 0.03$). in contrast, no difference was found between the two diagnostic groups in the levels of beta-thromboglobulin, prothrombin fragment 1+2, fibrinogen, and von Willebrand factor. No other association but **diagnosis** was found with thrombomodulin and sE-selectin. These findings suggest that endothelial dysfunction is an early event in AD patients.

L20 ANSWER 7 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2002006005 EMBASE Amyloid precursor protein in **platelets**: A peripheral marker for the **diagnosis** of sporadic AD. Padovani A.; Pastorino L.; Borroni B.; Colciaghi F.; Rozzini L.; Monastero R.; Perez J.; Pettenati C.; Mussi M.; Parrinello G.; Cottini E.; Lenzi G.L.; Trabucchi M.; Cattabeni F.; Di Luca M.. Dr. A. Padovani, Dipto. Scienze Mediche e Chirurgiche, Clinica Neurologica, Universita degli Studi di Brescia, 25125 Brescia, Italy. padovani@bshosp.osp.unibs.it. Neurology 57/12 (2243-2248) 26 Dec 2001.
Refs: 37.
ISSN: 0028-3878. CODEN: NEURAI. Pub. Country: United States. Language: English. Summary Language: English.

AB Background: An altered pattern of amyloid precursor protein (APP) forms consisting in a reduced ratio between the upper (130 kDa) and the lower (106 to 110 kDa) immunoreactivity bands has been described in **platelets** of patients with AD. Objective: To evaluate the sensitivity and the specificity of **platelet** APP forms' ratio (APPr) as a marker for AD. Methods: Eighty-five patients with probable AD and 95 control subjects (CON), including healthy individuals and neurologic patients, entered the study. **Platelet** APPr was evaluated by means of Western Blot analysis and immunostaining in the whole **platelet** homogenate, and calculated by the ratio between the optical density (OD) of the upper (130 kDa) and the lower (106 to 110 kDa) APP immunoreactive bands. Results: Mean APPr levels were decreased in AD patients (mean OD .+-. SD = 0.35 .+-. 0.18) compared with the CON group (mean OD .+-. SD = 0.92 .+-. 0.38) (DF 1, 178, $p < 0.0001$). Accuracy levels measured by Receiver Operating Curve analysis showed that a cut-off level of 0.57 resulted in a sensitivity of 88.2% and a specificity of 89.4%, with an area under the curve of 0.945. APPr levels were significantly associated with disease severity (mild AD versus moderate AD: $p < 0.0001$; moderate AD versus severe AD: $p < 0.05$). Conclusion: **Platelet** APPr allowed to differentiate AD from normal aging and other dementing disorders with high sensitivity and specificity. These findings suggest that **platelet** APPr may be of help as an adjunctive diagnostic tool in clinical practice.

L20 ANSWER 8 OF 47 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2001:337479 Document No.: PREV200100337479. Clinical correlates and drug treatment of residents with stroke in long-term care. Quilliam, Brian J.; Lapane, Kate L. (1). (1) Brown University, Providence, RI, 02912: Kate_Lapane@brown.edu USA. Stroke, (June, 2001) Vol. 32, No. 6, pp. 1385-1393. print. ISSN: 0039-2499. Language: English. Summary Language: English.

AB Background and Purpose: Stroke incidence increases with age, and stroke survivors often require nursing home placement. Characteristics of these residents and factors associated with the secondary drug prevention of stroke in nursing homes have yet to be explored. Methods: We used a population-based data set of all nursing home residents in 5 states (1992 to 1995). We identified 53 829 (20.4%) with a **diagnosis** of stroke on the Minimum Data Set assessment. We considered aspirin, dipyridamole, ticlopidine, or warfarin alone or in combination as secondary drug prevention. We used logistic regression modeling to identify independent predictors of drug treatment. Results: Sixty-seven percent of stroke survivors were not receiving drug therapy for stroke prevention. Among those treated, most received aspirin alone (16%) or

warfarin alone (10%). Independent predictors of drug treatment included comorbid conditions (eg, hypertension, atrial fibrillation, depression, **Alzheimer**'s disease, **dementia**, gastrointestinal bleeding, and peptic ulcer disease). Those over the age of 85 years were less likely to be treated than those 65 to 74 years of age (odds ratio (OR), 0.86; 95% confidence interval (CI), 0.82 to 0.91); black residents were less likely to be treated than whites (OR, 0.80; 95% CI, 0.75 to 0.85); and those with severe cognitive (OR, 0.63; 95% CI, 0.60 to 0.67) or physical impairment (OR, 0.69; 95% CI, 0.64 to 0.75) were also less likely to receive drug treatment. Conclusions: Stroke is highly prevalent in long-term care. Despite the increased risk of subsequent stroke in the elderly, many are not being treated. The choice to treat or not to treat may be influenced by age, comorbidity, race/ethnicity, and cognitive or physical functioning.

L20 ANSWER 9 OF 47 MEDLINE
2001698973 Document Number: 21532849. PubMed ID: 11675581. [Two case reports of cerebral autosomal dominant arteriophaty with subcortical infarctions and leukoencephalopathy (CADASIL)]. Arteriopatia cerebrale autosomica dominante con infarti sottocorticali e leucoencefalopatia (CADASIL). Pellicano S; Costa A; Terra L. (A.S.L. n. 5 - Crotone, Ospedale S. Giovanni di Dio, Divisione di Malattie Infettive, Universita di Pavia.) MINERVA MEDICA, (2001 Oct) 92 (5) 381-4. Journal code: 0400732. ISSN: 0026-4806. Pub. country: Italy. Language: Italian.

AB Cerebral autosomal dominant arteriopathy with subcortical infarctions and leukoencephalopathy (CADASIL) was first reported in European families and since 1993 it has been observed in America, Africa and Asia, suggesting that today the disease probably still remains largely underdiagnosed. CADASIL appears to be essentially a disorder of the arteries linked to single missense mutations in the Notch3 gene locus on chromosome 19; the aberrant dimerisation of Notch3, due to abnormal disulphide bridging with another Notch3 molecule or with another protein, may be involved in the pathogenesis of the disorder. It is characterized by recurrent stroke episodes and focal neurologic deficits progressing to pseudobulbar palsy and **dementia**, caused by multiple lacunar infarctions with ischemic and diffuse white matter abnormalities on neuroimaging. Migraine with aura, epileptic seizures and affective disorders are frequent additional symptoms of CADASIL. It is usually observed in the 3rd decade, but some individuals remain asymptomatic close to the age of 60. MRI displays a marked leukoencephalopathy in affected individuals as early as in the age of 20. The authors emphasize the role of a direct DNA test for gene mutation to make a differential **diagnosis** between CADASIL and other forms of vascular leukoencephalopathy as **Alzheimer**'s **dementia**, multiple sclerosis and Binswanger's subcortical arteriopathic encephalopathy where CADASIL's arteriopathy is characterized by major alterations of vascular smooth muscle cells and the presence of specific granular osmiophilic deposits.

L20 ANSWER 10 OF 47 MEDLINE
2000467783 Document Number: 20475371. PubMed ID: 11021167. [Guidelines for the treatment of **dementias**]. Linee guida per la terapia delle demenze. Trabucchi M; Bianchetti A. RECENTI PROGRESSI IN MEDICINA, (2000 Sep) 91 (9) 444-9. Ref: 49. Journal code: 0401271. ISSN: 0034-1193. Pub. country: Italy. Language: Italian.

L20 ANSWER 11 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
2000:847503 The Genuine Article (R) Number: 370NM. Anti-inflammatory drugs protect against **Alzheimer** disease at low doses. Broe G A (Reprint); Grayson D A; Creasey H M; Waite L M; Casey B J; Bennett H P; Brooks W S; Halliday G M. PRINCE WALES MED RES INST, HIGH ST, RANDWICK, NSW 2031, AUSTRALIA (Reprint); UNIV SYDNEY, CTR EDUC & RES AGING, SYDNEY, NSW 2006, AUSTRALIA; CONCORD HOSP, DEPT MED, CONCORD, NSW, AUSTRALIA.

ARCHIVES OF NEUROLOGY (NOV 2000) Vol. 57, No. 11, pp. 1586-1591. Publisher: AMER MEDICAL ASSOC. 515 N STATE ST, CHICAGO, IL 60610. ISSN: 0003-9942. Pub. country: AUSTRALIA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Context: Anti-inflammatory medications have an inverse association with **Alzheimer** disease (AD).

Objectives: To examine at what doses this anti-inflammatory drug effect occurs and whether other medications and/or International Classification of Diseases, Ninth Revision, Clinical Modification **diagnoses** affect the association.

Design: Subjects 75 years and older from a random population sample were classified by consensus using International Classification of Diseases, Ninth Revision, Clinical Modification **diagnoses**. Drug associations with different types of **dementia** with and without the International Classification of Diseases, Ninth Revision, Clinical Modification **diagnoses** as well as dosage data were analyzed.

Setting: The Centre for Education and Research on Aging, Concord Hospital, Concord, Australia.

Patients: The Sydney Older Persons Study recruited 647 subjects (average age, 81 years). A total of 163 patients were given **diagnoses** placing them in different **dementia** categories and were compared with 373 control subjects. Of the patients with **dementia**, 78 had AD without vascular **dementia**, 45 had vascular **dementia** (permissive of other **dementia diagnoses**), and 40 had other **dementia diagnoses** (without AD or vascular **dementia**).

Main Outcome Measures: Fifty drugs or drug groups were subjected to a 2 (drug used vs drug not used) x 4 (**dementia** and control groups) chi (2) analysis. Drugs with inverse associations were identified and potential confounders (logistic regression) and dosage data (exact small sample 1-tailed tests) analyzed.

Results: As expected, there was an inverse association between nonsteroidal anti-inflammatory drugs and aspirin (and unexpectedly angiotensin-converting enzyme inhibitors) and AD. This association was not observed with vascular **dementia** or any other **diagnoses** . Analysis showed no evidence for a dosage effect, ie, responses were equivalent for low and high doses.

Conclusions: This study does not support a high-dose anti-inflammatory action of nonsteroidal anti-inflammatory drugs or aspirin in AD. Potential mechanisms for the beneficial effects of these medications are discussed.

L20 ANSWER 12 OF 47 MEDLINE

1999365440 Document Number: 99365440. PubMed ID: 10436098. Increased thromboxane biosynthesis is associated with poststroke **dementia**.
van Kooten F; Ciabattoni G; Koudstaal P J; Grobbee D E; Kluft C; Patrono C. (Department of Neurology, University Hospital Rotterdam, Rotterdam, the Netherlands.. vankooten@neuro.fgg.eur.nl) . STROKE, (1999 Aug) 30 (8) 1542-7. Journal code: 0235266. ISSN: 0039-2499. Pub. country: United States. Language: English.

AB BACKGROUND AND PURPOSE: It has been suggested that daily intake of aspirin is associated with a reduction of cognitive decline, both in normal and in demented subjects, but the mechanism is unclear. We have therefore studied the relationship between thromboxane (TX) A(2) biosynthesis, as reflected by the urinary excretion of 11-dehydro-TXB(2), and the presence of **dementia** in patients after acute stroke. METHODS: Patients from the Rotterdam Stroke Databank were screened for **dementia** between 3 and 9 months after stroke. Patients had a full neurological examination, neuropsychological screening, and, if indicated, extensive neuropsychological examination. Criteria used for the **diagnosis** of **dementia** were from the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (Revised). Urine samples were taken at the time of screening. Urinary 11-dehydro-TXB(2) was measured by means of

a previously validated radioimmunoassay. RESULTS: **Dementia** was diagnosed in 71 patients, and urine samples were available for 62. Median value (range) of 11-dehydro-TXB(2) was 399 (89 to 2105) pmol/mmol creatinine for demented patients versus 273 (80 to 1957) for 69 controls with stroke but without **dementia** (P=0.013). No difference was found between 44 patients with vascular **dementia**, 404 (89 to 2105) pmol/mmol creatinine, and 18 patients with **Alzheimer**'s disease plus cerebrovascular disease, 399 (96 to 1467) pmol/mmol creatinine (P=0.68). In a stepwise logistic regression analysis, in which possible confounders such as use of antiplatelet medication, cardiovascular risk factors, and type of stroke were taken into account, increased urinary excretion of 11-dehydro-TXB(2) remained independently related to the presence of **dementia** (OR 1.12, 95% CI 1.03 to 1.22 per 100 pmol/mmol creatinine). The difference in metabolite excretion rates between demented and nondemented patients was most prominent within the subgroup of ischemic stroke patients who received aspirin (P<0.01). CONCLUSIONS: Increased thromboxane biosynthesis in the chronic phase after stroke is associated with the presence of but not the type of poststroke **dementia**. It is particularly apparent in patients on aspirin, thereby suggesting the involvement of extraplatelet sources of TXA(2) production in this setting.

L20 ANSWER 13 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)

1999:569880 The Genuine Article (R) Number: 217BL. Clinical and neurobiological correlates of DXS1047 genotype in **Alzheimer**'s disease. Zubenko G S (Reprint); Hughes H B; Stiffler J S. WESTERN PSYCHIAT INST & CLIN, ROOM E-1230, 3811 OHARA ST, PITTSBURGH, PA 15213 (Reprint); UNIV PITTSBURGH, SCH MED, DEPT PSYCHIAT, PITTSBURGH, PA; CARNEGIE MELLON UNIV, MELLON COLL SCI, DEPT BIOL SCI, PITTSBURGH, PA 15213 . BIOLOGICAL PSYCHIATRY (15 JUL 1999) Vol. 46, No. 2, pp. 173-181. Publisher: ELSEVIER SCIENCE INC. 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010. ISSN: 0006-3223. Pub. country: USA. Language: English. *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Background: The goal of the current study was to explore the clinical, neuropathological, and neurochemical correlates of the DXS1047 202bp allele in a group of 50 autopsy-confirmed cases of **Alzheimer**'s disease (AD) who lacked other concomitant brain diseases. We previously published the results of a genome survey for novel risk loci for typical-onset (greater than or equal to 60 years) AD conducted at 10cM resolution (Zubenko et al 1998a, b). This survey detected associations of alleles at six microsatellite loci with AD, including the 202bp allele of the DXS1047 locus that resides within Xq25 on the human cytogenetic map,

Methods: Clinical assessments were performed as part of a longitudinal study of AD and related disorders. Autopsies were performed using standardized methods and the resulting **diagnoses** were made according to established criteria. Genotyping, morphometry, and neurochemical analyses were performed using postmortem brain tissue.

Results: Patients with AD who carried the DXS1047 202bp allele manifested cortical norepinephrine levels that ranged from 2.1 to 3.6 times the corresponding values for noncarriers (p = .002), controlling for the potential effects of gender; age at symptomatic onset or death, and postmortem interval. In contrast, carriers tended to have lower cortical levels of dopamine (p = .10).

Conclusions: These findings support the results of our previous genome survey and suggest that the DXS1047 locus, or a locus in close proximity, modulates biological variables relevant to the pathophysiology of AD. In addition to providing insights into the clinical biology of AD, the characterization of biologically meaningful subtypes, including genotypic subtypes associated with particular neurobiological derangements, may be important to the advancement of experimental therapeutics in AD. (C) 1999 Society of Biological Psychiatry.

L20 ANSWER 14 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
1998:880079 The Genuine Article (R) Number: 138QJ. The efficacy of Ginkgo biloba on cognitive function in **Alzheimer** disease. Oken B S (Reprint); Storzbach D M; Kaye J A. OREGON HLTH SCI UNIV, DEPT NEUROL, CR120, 3181 SW SAM JACKSON PK RD, PORTLAND, OR 97201 (Reprint); OREGON HLTH SCI UNIV, CTR RES OCCUPAT & ENVIRONM TOXICOL, PORTLAND, OR 97201; PORTLAND VET AFFAIRS MED CTR, PORTLAND, OR. ARCHIVES OF NEUROLOGY (NOV 1998) Vol. 55, No. 11, pp. 1409-1415. Publisher: AMER MEDICAL ASSOC. 515 N STATE ST, CHICAGO, IL 60610. ISSN: 0003-9942. Pub. country: USA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective: To determine the effect of treatment with Ginkgo biloba extract on objective measures of cognitive function in patients with **Alzheimer** disease (AD) based on formal review of the current literature.

Methods: An attempt was made to identify all English and non-English-language articles in which G biloba extract was given to subjects with **dementia** or cognitive impairment. Inclusion criteria for the meta-analysis were (1) sufficiently characterized patients such that it was clearly stated there was a **diagnosis** of AD by either Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition, or National Institute of Neurological Disorders and Stroke-**Alzheimer**'s Disease and Related Disorders Association criteria, or there was enough clinical detail to determine this by our review; (2) clearly stated study exclusion criteria, ie, those studies that did not have stated exclusions for depression, other neurologic disease, and central nervous system-active medications were excluded; (3) use of standardized ginkgo extract in any stated dose; (4) randomized, placebo-controlled and double-blind study design; (5) at least 1 outcome measure was an objective assessment of cognitive function; and (6) sufficient statistical information to allow for meta-analysis.

Results: Of more than 50 articles identified, the overwhelming majority did not meet inclusion criteria, primarily because of lack of clear **diagnoses** of **dementia** and AD. Only 4 studies met all inclusion criteria. In total there were 212 subjects in each of the placebo and ginkgo treatment groups. Overall there was a significant effect size of 0.40 (P<.0001). This modest effect size translated into a 3% difference in the **Alzheimer** Disease Assessment Scale-cognitive subtest.

Conclusions: Based on a quantitative analysis of the literature there is a small but significant effect of 3- to 6-month treatment with 120 to 240 mg of G biloba extract an objective measures of cognitive function in ED. The drug has not had significant adverse effects in formal clinical trials but there are 2 case reports of bleeding complications. In AD, there are limited and inconsistent data that preclude determining if there are effects on noncognitive behavioral and functional measures as well as on clinician's global rating scales. Further research in the area will need to determine if there are functional improvements and to determine the best dosage. Additional research will be needed to define which ingredients in the ginkgo extract are producing its effect in individuals with AD.

L20 ANSWER 15 OF 47 MEDLINE
1998410829 Document Number: 98410829. PubMed ID: 9740113. Differential level of **platelet** amyloid beta precursor protein isoforms: an early marker for **Alzheimer** disease. Di Luca M; Pastorino L; Bianchetti A; Perez J; Vignolo L A; Lenzi G L; Trabucchi M; Cattabeni F; Padovani A. (Institute of Pharmacological Sciences, University of Milan, Milano, Italy.. diluca@imiucca.csi.unimi.it) . ARCHIVES OF NEUROLOGY, (1998 Sep) 55 (9) 1195-200. Journal code: 0372436. ISSN: 0003-9942. Pub. country: United States. Language: English.

AB OBJECTIVE: To determine whether a differential level of **platelet**

amyloid beta precursor protein (APP) isoforms is specifically related to **Alzheimer** disease (AD) and whether it shows a correlation with the progression of clinical symptoms. DESIGN: After subjects were grouped according to **diagnosis** and severity of **dementia**, APP isoform levels in **platelets** were compared. SETTING: University medical centers. PATIENTS: Thirty-two patients who fulfilled diagnostic criteria for probable AD, 25 age-matched control subjects, and 16 patients with non-AD **dementia**. MAIN OUTCOME MEASURE: The levels of APP isoforms were evaluated by means of Western blot analysis and immunostaining of whole **platelets**. Messenger RNAs for APP transcripts were also evaluated by means of reverse transcriptase polymerase chain reaction. RESULTS: The ratio between the intensity of the 130-kd and 106- to 110-kd APP isoforms was significantly lower in the AD group (0.31 +/- 0.15, mean +/- SD) compared with both controls (0.84 +/- 0.2) and non-AD subjects (0.97 +/- 0.4). The ratio of **platelet** APP isoforms in patients with AD grouped by Clinical Diagnostic Rating score significantly correlated with the severity of the disease (Pearson correlation coefficient, followed by Bonferroni correction, P = .01). Reverse transcriptase polymerase chain reaction experiments showed that APP transcripts in all experimental groups were equally expressed. CONCLUSIONS: The pattern of **platelet** APP isoforms is specifically altered in patients with AD. In addition, the alteration of **platelet** APP isoforms shows a positive correlation with the progression of clinical symptoms, supporting the possibility to consider this peripheral parameter as a marker of progression of the disease. These alterations are not related to abnormalities of APP isoforms messenger RNAs in **platelets**.

L20 ANSWER 16 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)

1998:725428 The Genuine Article (R) Number: 120PH. Biomedical applications of maghemite ferrofluid. Halbreich A (Reprint); Roger J; Pons J N; Geldwerth D; DaSilva M F; Roudier M; Bacri J C. UNIV PARIS 06, LAB MILIEUX DESORDONNES & HETEROGENES, CASE 78, 4 PL JUSSIEU, F-75252 PARIS 05, FRANCE (Reprint); UNIV PARIS 06, LAB LIQUIDES ION & INTERFACES CHARGES, CASE 63, F-75252 PARIS 05, FRANCE; IBPC, F-75005 PARIS, FRANCE; UNIV BRASILIA, DEPT FIS, BR-70910900 BRASILIA, DF, BRAZIL; HOP CHARLES RICHET, SERV GERONTOL CLIN, F-95540 VILLIERS LE BEL, FRANCE. BIOCHIMIE (MAY-JUN 1998) Vol. 80, No. 5-6, pp. 379-390. Publisher: EDITIONS SCIENTIFIQUES MEDICALES ELSEVIER . 23 RUE LINOIS, 75724 PARIS CEDEX 15, FRANCE. ISSN: 0300-9084. Pub. country: FRANCE; BRAZIL. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The use of cell-targeted ferrofluid in the characterization of modifications of cell membranes is reviewed. Maghemite ferrofluid was synthesized by the Massart method, complexed with dimercaptosuccinic acid (FF). Cell targeting by FF was developed by coupling FF to various biological effecters such as antibodies, lectins, etc, which enabled magnetic cell sorting. Modifications in erythrocyte membranes were studied using FF bound to recombinant human annexin V (AN;FF) which is very sensitive, compared to other Anx-based reagents, in the early detection of phosphatidylserine (PS) exposition on the outer leaflet of the plasma membrane. Thus PS exposition on mouse RBC was detected already after a 24-h storage at 4 degrees C and, transiently, 24 h after their infection by Plasmodium parasites, at which time the parasites are Still confined to the liver, thus leading to the recruitment of young RBC and the accumulation of a species, intermediate between reticulocytes and erythrocytes, and the actual RBC target of plasmodial invasion. AnxFF revealed PS exposition on RBC from sickle cell anemia patients, following various inflammations and already after 20 days of human blood storage under blood bank conditions. Such a sensitive detection should be similar to that of macrophages which recognize exposed PS on cells and bring about the latter's elimination from the circulation. AnxFF binding determination was combined with that of cell electrophoretic mobility, glycerol

resistance and filterability to characterize RBC membrane modifications in **Alzheimer**'s disease patients which suggested a continuous damage and regeneration in RBC of these patients. A logistic analysis suggested that several three-parameter combinations could permit **diagnosis** of **Alzheimer**'s disease with up to 95% accuracy. THP1 cells and macrophages, derived themselves by incubation with retinoic acid, were bound to FF and placed in a radio frequency alternating magnetic field. Magnetocytolysis was associated with FF attachment to the cells without damage to non-bound cells and without heating of the surrounding solution ((C) Societe francaise de biochimie et biologie moleculaire / Elsevier, Paris).

L20 ANSWER 17 OF 47 CAPLUS COPYRIGHT 2003 ACS
1997:329345 Document No. 126:304914 Assay for the **diagnosis** of **dementia**. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co., Ltd., Israel; Shinitzky, Meir; Deckmann, Michael). PCT Int. Appl. WO 9713152 A1 19970410, 21 pp. DESIGNATED STATES: W: AU, BR, CA, IL, JP, US; RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1996-IL118 19960926. PRIORITY: IL 1995-115465 19950929.
AB An assay for the **diagnosis** of multi-infarct **dementia** and **dementia** of the **Alzheimer** type in an individual is provided. The method is an immunoassay for detn. of a 75 kD **platelet** protein and/or the **platelet** assocd. antibodies against the 75 kD **platelet** protein in the blood sample. A level higher than that of a control sample indicates that said individual has a high likelihood of having multi-infarct **dementia** or **dementia** of the **Alzheimer** type.

L20 ANSWER 18 OF 47 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1997:469673 Document No.: PREV199799768876. APP isoforms in **platelets** : A peripheral marker for **Alzheimer** disease. Pastorino, L. (1); Di Luca, M.; Perez, J.; Bianchetti, A.; Vignolo, L. A.; Lenzi, G. L.; Trabucchi, M.; Cattebeni, F.; Padovani, A.. (1) Inst. Pharmacol. Sci., Univ. Milano, Milano Italy. Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 536. Meeting Info.: 27th Annual Meeting of the Society for Neuroscience, Part 1 New Orleans, Louisiana, USA October 25-30, 1997 ISSN: 0190-5295. Language: English.

L20 ANSWER 19 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
96:196913 The Genuine Article (R) Number: TY356. PROSPECTIVE-STUDY OF INCREASED **PLATELET** MEMBRANE FLUIDITY AS A RISK FACTOR FOR **ALZHEIMERS**-DISEASE - RESULTS AT 5 YEARS. ZUBENKO G S (Reprint); TEPLY I; WINWOOD E; HUFF F J; MOOSSY J; SUNDERLAND T; MARTINEZ A J. WESTERN PSYCHIAT INST & CLIN, 3811 OHARA ST, RM E-1230, PITTSBURGH, PA, 15213 (Reprint); UNIV PITTSBURGH, SCH MED, DEPT PSYCHIAT, PITTSBURGH, PA, 00000; UNIV PITTSBURGH, SCH MED, DEPT PATHOL NEUROPATHOL, PITTSBURGH, PA, 00000; UNIV MED & DENT NEW JERSEY, PISCATAWAY, NJ, 08854; NIMH, CLIN SCI LAB, SECT GERIATR PSYCHIAT, BETHESDA, MD, 20892. AMERICAN JOURNAL OF PSYCHIATRY (MAR 1996) Vol. 153, No. 3, pp. 420-423. ISSN: 0002-953X. Pub. country: USA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Objective: The primary goal of this study was to evaluate increased **platelet** membrane fluidity as a putative risk factor for **Alzheimer**'s disease. Method: This report describes the initial results of a prospective, longitudinal study of 330 initially asymptomatic, first-degree relatives of probands with **Alzheimer**'s disease. Results: Five incident cases of **Alzheimer**'s disease were detected during the first 1,582 subject-years of the follow-up period. The age-specific incidence of **Alzheimer**'s disease was several-fold higher than corresponding figures that were obtained in two prospective community studies. Most important, both age and increased

platelet membrane fluidity made significant independent contributions to the risk of developing **Alzheimer**'s disease. Conclusions: These results validate age and a family history of **Alzheimer**'s disease as risk factors for this disorder and provide the first prospective evidence of increased **platelet** membrane fluidity as a biological risk factor for **Alzheimer**'s disease.

L20 ANSWER 20 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
96:338596 The Genuine Article (R) Number: UG525. **PLATELETS** FROM PATIENTS WITH **ALZHEIMERS**-DISEASE OR OTHER **DEMENTIAS** EXHIBIT DISEASE-SPECIFIC AND APOLIPOPROTEIN-E CORRELATABLE DEFECTS. DAVIES T A (Reprint); LONG H J; RATHBUN W H; SGRO K R; TIBBLES H; SMITH S J; SEETOO K F; MCMENAMIN M E; JOHNSON R; WELLS J M; LEVESQUE C; FINE R E; SIMONS E R. BOSTON UNIV, SCH MED, DEPT BIOCHEM, BOSTON, MA, 02118 (Reprint); EDITH NOURSE ROGERS VET ADM HOSP, BEDFORD, MA, 01730; BOSTON CITY HOSP, NEUROL UNIT, BOSTON, MA, 02118. AMYLOID-INTERNATIONAL JOURNAL OF EXPERIMENTAL AND CLINICAL INVESTIGATION (MAR 1996) Vol. 3, No. 1, pp. 13-19. ISSN: 1350-6129. Pub. country: USA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **Platelets** carry over 95% of the circulating **Alzheimer**'s beta-amyloid precursor protein (A beta PP), and release soluble and hydrophobic proteolytic fragments of A beta PP upon activation. These cells may be the source of cerebrovascular amyloid peptides, a part of **Alzheimer**'s disease (AD) pathology. Our previous studies showed that **platelets** from patients with advanced AD exhibit both signal transduction (hyperacidification) and A beta PP processing defects. Here, we show further that a similar hyperacidification also exists in patients with Pick's disease (a **dementia** with AD-like symptoms but a different amyloid pathology) or Down syndrome (trisomy and hence overproduction of A beta PP), while the A beta PP processing defect and consequent A beta PP retention on the membrane is absent and is thus likely to he AD-specific. The hyperacidification defect correlates with all three **dementias** and with the presence of apolipoprotein E4 which has been implicated as a risk factor for AD.

L20 ANSWER 21 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
95237234 EMBASE Document No.: 1995237234. Importance of **platelet** functions in **Alzheimer**'s disease. Hasitz M.; Racz Z.; Nagy A.; Lipcsey A.. St. John Hospital, Neuropsychiatric Department, Diosarok u. 1,Budapest 1125, Hungary. Archives of Gerontology and Geriatrics 21/1 (53-61) 1995.
ISSN: 0167-4943. CODEN: AGGEDL. Pub. Country: Ireland. Language: English. Summary Language: English.

AB The function of **platelets** of patients with **Alzheimer**'s disease has been characterized. The shape of **platelets** is more spherical and the initial rate of **platelet** aggregation caused by different agonists measured in plasma is faster in the case of **Alzheimer**'s disease and senile **dementia** of the **Alzheimer**-type than that of other demented patients: multi infarct **dementia** and probable vascular **dementia**. Although fast aggregation is characteristic of activated **platelets** but the **Alzheimer platelets** are not hypersensitive. The activation of **Alzheimer platelets** makes the velocity of ADP induced aggregation slower and it is different than the behaviour of normal activated **platelets**. The shape-associated parameter and the initial rate of 50 .mu.M ADP-induced aggregation of **platelets** non-activated and activated by cytochrome C are recommended for establishing the **diagnosis** of **Alzheimer**'s disease and senile **dementia** of the **Alzheimer**-type. The evaluation of these parameters has been discussed. The cytochrome C may be useful to normalize the function of **Alzheimer platelets** in plasma, not only in vitro.

L20 ANSWER 22 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
94:769886 The Genuine Article (R) Number: PU842. GABA-TRANSAMINASE IN BRAIN AND BLOOD-**PLATELETS** - BASIC AND CLINICAL ASPECTS. SHERIF F M (Reprint). UNIV MED SCI, DEPT PHARMACOL, POB 82757, TRIPOLI, LIBYA (Reprint). PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY (DEC 1994) Vol. 18, No. 8, pp. 1219-1233. ISSN: 0278-5846. Pub. country: LIBYA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB 1. Several lines of evidence suggest that the major inhibitory neuro-transmitter, gamma-aminobutyric acid (GABA) is involved, both directly and indirectly, in the pathogensis of certain neurological, and psychiatric disorders.

2. The main enzyme responsible for GABA catabolism is gamma-aminobutyrate aminotransferase (GABA-T). Inhibition of this enzyme produces a considerable elevation of brain GABA concentrations, and such elevation has been correlated with many pharmacological effects.

3. There seems to be that, as is discussed below, GABA-T activity in the brain and/or blood **platelets** is related to some neuro-psychiatric disorders such as alcoholism, epilepsy and **Alzheimer**'s disease.

4. GABA-T has been identified in the blood **platelets** with similar characteristics to those of brain GABA-T. In this way, studies on GABA-T activity in neuro-psychiatric disorders could be performed to understand, **diagnosis** and treat GABA-related disorders of the central nervous system (CNS).

L20 ANSWER 23 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
94:494923 The Genuine Article (R) Number: PB130. BETA-AMYLOID PROTEIN IMMUNOREACTIVITY IN SKIN IS NOT A RELIABLE MARKER OF **ALZHEIMERS** -DISEASE - AN AUTOPSY-CONTROLLED STUDY. HEINONEN O (Reprint); SOININEN H; SYRJANEN S; NEITTAANMAKI H; PALJARVI L; KOSUNEN O; SYRJANEN K; RIEKKINEN P . UNIV KUOPIO, DEPT NEUROL, POB 1627, SF-70211 KUOPIO, FINLAND (Reprint); UNIV KUOPIO, DEPT DERMATOL, SF-70211 KUOPIO, FINLAND; UNIV KUOPIO, AI VIRTANEN INST, SF-70211 KUOPIO, FINLAND. ARCHIVES OF NEUROLOGY (AUG 1994) Vol. 51, No. 8, pp. 799-804. ISSN: 0003-9942. Pub. country: FINLAND. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objectives: As a possible diagnostic marker for **Alzheimer**'s disease (AD), we investigated beta-amyloid protein (beta/A4) immunoreactivity in skin. Furthermore, we studied the presence of beta-amyloid precursor protein 695 immunoreactivity in skin.

Design: Lifetime skin biopsy specimens were stained for beta/A4 and beta-amyloid precursor protein 695. The follow-up period was 12 months. We determined the correlation between beta/A4 immunoreactivity in skin and brain in patients with a neuropathologic **diagnosis**.

Setting: All patients with **dementia** were hospitalized; most of them had moderate to severe **dementia**. Aged nondemented controls were residents of a nursing home. The Down's syndrome (DS) group included both hospitalized and ambulatory patients. Young nondemented controls were medical students or staff members who volunteered for the study.

Patients and Other Participants: The study included a total of 111 subjects. Thirty-five patients had probable AD, nine had possible AD, 15 had multiinfarct **dementia**, one had idiopathic Parkinson's disease, and one had Parkinson's disease and possible AD. There were also 19 elderly nondemented controls, 23 patients with DS, and eight young nondemented controls.

Main Outcome Measures: Immunohistochemical detec tion of beta/A4 in skin and correlation to the **diagnosis** of AD.

Results: immunopositivity for beta/A4 antibody was present in and around the endothelium of dermal blood vessels in a proportion of patients

with AD and multi-infarct **dementia** as well as elderly controls. The patients with sporadic AD displayed beta/A4 immunoreactivity significantly more frequently than did patients with familial AD, patients with multi-infarct **dementia**, and controls. The beta/A4 immunopositivity in skin was rare in the patients with DS and not present in young controls. Instead, 48% of patients with DS but none of other groups had beta-amyloid precursor protein 695 immunoreactivity in skin. Only four (31%) of 13 patients with neuropathologically confirmed AD had shown endothelial beta/A4 immunopositivity in skin biopsy specimens while alive.

Conclusion: Our results do not support beta/A4 as a diagnostic marker for AD.

L20 ANSWER 24 OF 47 MEDLINE

94318776 Document Number: 94318776. PubMed ID: 8043706. **Platelet** monoamine oxidase B activity and vitamin B12 in **dementia**. Fischer P; Gotz M E; Ellinger B; Streifler M; Riederer P; Danielczyk W. (Psychiatric Clinic, University of Vienna, Austria.) BIOLOGICAL PSYCHIATRY, (1994 May 15) 35 (10) 772-4. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

AB The activity of **platelet** monoamine oxidase B (MAO-B) was highly correlated with the severity of **dementia** in 39 patients suffering from probable **dementia** of the **Alzheimer** type and in 18 age-matched controls. There was no association between a low vitamin B12 level and high MAO-B activity in our sample of patients, who are living in a geriatric hospital where the balanced nutrition of inpatients is controlled by diet assistants.

L20 ANSWER 25 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

94111858 EMBASE Document No.: 1994111858. A 78-year-old man with progressive gait disturbance, dysphagia, and **dementia**. Matsuda K.; Hatori K.; Ohtani H.; Mori H.; Suda K.; Imai H.; Mizuno Y.. Department of Neurology, University School of Medicine, 2-1-1 Hongo,Bunkyo, Tokyo 113, Japan. Brain and Nerve 46/3 (297-305) 1994. ISSN: 0006-8969. CODEN: NOTOA6. Pub. Country: Japan. Language: Japanese. Summary Language: Japanese; English.

AB We report a 78-year-old man with progressive gait disturbance, **dementia**, and dysphagia. He was apparently well until 75 years of age in 1989 when he had an insidious onset of gait disturbance. In October of 1991, he was treated with levodopa and amantadine HCL in another hospital, but he developed visual hallucination right after these medications, and the drugs were discontinued. He also developed difficulty in swallowing with frequent aspiration pneumonia. He was admitted to our hospital on January 13, 1992. On admission, the patient was chronically ill Japanese man; his blood pressure was 118/70 mmHg, body temperature 35.4.degree.C, and heart rate 72 and regular. No anemia or jaundice was noted; lungs were clear and no heart murmur was audible. The abdomen was flat but rigid to palpation without tenderness; no organomegaly was noted. On neurologic examination, he was alert but disoriented to all spheres; he was apparently demented and the score of the mini-mental test was 11. He did not appear to have aphasia or apraxia. Cranial nerves appeared intact, but he had a mask-like face and a slight limitation in the upward gaze; his voice was small. He was unable to stand or walk; he showed marked akinesia and moderate rigidity in his neck and the trunk. Deep reflexes were generally elicited normally or slightly weakly. Plantar response was extensor on the left and flexor on the right. No grasp reflex was present. Sensory examination showed questionable loss of touch in the glove-and -stocking distribution. He developed fever and a pneumonic shadow in his right lung field; he was treated with antibiotics and intravenous fluid. His condition deteriorated and an intratracheal tube was inserted on January 17, 1992. He suddenly vomited bloody materials on January 19, and blood **platelet** count decreased to 35,000/.mu.l. His pneumonia

further deteriorated on January 24, and he developed cardiopulmonary arrest on January 26, 1992. The patient was discussed in a neurological CPC. Because of poor information in his past medical record, differential **diagnosis** was difficult, but the chief discussant arrived at a conclusion that the patient had striato-nigral degeneration, because he had levodopa-non-responsive parkinsonism and **dementia**.
Post-mortem examination revealed marked neuronal loss in the substantia nigra pars compacta with Lewy bodies in the remaining neurons. Neuronal loss was also observed in the locus coeruleus, dorsal motor nucleus of the vagal nerve, and the basal nucleus of Meynert. Lewy bodies were also seen in the cortical areas particularly in the insular cortex. In the cortical areas, numerous senile plaques, mostly diffuse plaques, were seen, however, **Alzheimer**'s neurofibrillary tangles were only rarely seen in the hippocampal neurons. It was concluded that the patient had the so-called diffuse Lewy body disease. This entity is unique in that the condition is characterized clinically by **dementia** and parkinsonism, and pathologically by marked neuronal loss in the substantia nigra, and the presence of Lewy bodies not only in the brain stem nuclei but also in the cerebral cortex. Another interesting feature is the marked senile change characterized by senile plaques not associated with the tangle formation except for hippocampal neurons. Whether this is a unique independent nosological entity or it is a form of Parkinson's disease associated with **dementia** awaits further investigations.

L20 ANSWER 26 OF 47 MEDLINE
95086098 Document Number: 95086098. PubMed ID: 7993928. **Platelet** MAO-B activity as a marker of behavioural characteristics in **dementia** disorders. Parnetti L; Reboldi G P; Santucci C; Santucci A; Gaiti A; Brunetti M; Cecchetti R; Senin U. (Dipartimento di Medicina Clinica, University of Perugia, Italy.) AGING, (1994 Jun) 6 (3) 201-7. Journal code: 9102503. ISSN: 0394-9532. Pub. country: Italy. Language: English.

AB Both low and high **platelet** MAO-B (pMAO-B) activity is considered an indicator of increased vulnerability in psychopathology. How the activity of this peripheral enzyme can be linked with the sophisticated functions of the central nervous system (CNS) is not clear; in man, evidence exists that the genetic mechanisms determining the size or capacity of the central serotonin system are common to **platelet** and brain MAO. In the present study pMAO-B activity was evaluated in demented patients suffering from early-onset **Alzheimer**'s disease (AD), late-onset **Alzheimer**'s disease (SDAT), vascular **dementia** (VD), and controls. In these **dementia** categories, the relationship between pMAO-B activity and clinical features, and between pMAO-B activity and cerebrospinal fluid (CSF) monoamine metabolites (3-methoxy-4-hydroxyphenyl-glycol, MHPG; 5-hydroxy-indoleacetic acid, 5-HIAA; homovanillic acid, HVA) was also investigated. pMAO-B activity was significantly higher in SDAT patients, compared to controls and AD. Age, as covariate, failed to show any significant effect, and no association was found between pMAO-B activity and CSF monoamine metabolites. The correlation analysis between pMAO-B and neuropsychological scores showed a highly significant positive relationship with GBS-emotional impairment ($N = 40$, $r = 0.72$, $p < 0.01$) in the SDAT group. This result suggests the importance of **platelet** MAO-B activity as biological marker also in old-age **dementias**, namely senile **dementia** of **Alzheimer** type, where the increased activity of this enzyme might constitute a marker for vulnerability toward behavioural disturbance, i.e., emotional deterioration.

L20 ANSWER 27 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
94:175654 The Genuine Article (R) Number: MZ294. BLOOD MARKERS IN **ALZHEIMER**-DISEASE - SUBNORMAL ACETYLCHOLINESTERASE AND

BUTYRYLCHOLINESTERASE IN LYMPHOCYTES AND ERYTHROCYTES. INESTROSA N C (Reprint); ALARCON R; ARRIAGADA J; DONOSO A; ALVAREZ J; CAMPOS E O. PONTIFICIA UNIV CATOLICA CHILE, FAC CIENCIAS BIOL, DEPT BIOL CELULAR & MOLEC, ALAMEDA 340, POB 114-D, SANTIAGO, CHILE (Reprint); UNIV CHILE, HOSP CLIN, SERV NEUROL & NEUROCIRUGIA, SANTIAGO, CHILE. JOURNAL OF THE NEUROLOGICAL SCIENCES (MAR 1994) Vol. 122, No. 1, pp. 1-5. ISSN: 0022-510X . Pub. country: CHILE. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In patients with the clinical **diagnosis** of **Alzheimer** disease (AD), we searched for systemic changes in components of the blood as a diagnostic tool. The acetylcholine-related enzymes acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE) were measured in plasma, erythrocytes, **platelets** and lymphocytes. Results did not show a general effect; notwithstanding, specific cell types presented alterations either in AChE or BuChE but not in both enzymatic activities. In AD patients, AChE of lymphocytes was reduced by 60% compared with the age-matched controls. However, when patients were divided, the sporadic but not the familial subgroup exhibited a significant reduction. In erythrocytes the BuChE activity was reduced by 45% in sporadic AD. The molecular forms of the lymphocyte AChE were characterized by velocity sedimentation. Both globular forms were subnormal, more so the tetrameric G(4) AChE form than the G(2) form.

L20 ANSWER 28 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
94038048 EMBASE Document No.: 1994038048. Changes of lymphocyte beta-adrenergic receptors and **platelet** serotonin reuptake sites in aging and **dementias**. Rozza A.; Scavini C.; Steardo L.; Guaita A.; Favalli L.; Racagni G.; Brunello N.. Institute of Pharmacology, University of Pavia, Via Taramelli 14,27100 Pavia, Italy. Human Psychopharmacology 8/5 (303-310) 1993.
ISSN: 0885-6222. CODEN: HUPSEC. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB We have studied the kinetic constants of .beta.-adrenergic receptors in lymphocytes and of serotonin uptake sites in **platelets** from adult and elderly volunteers and from aged patients with a **diagnosis** of multi-infarct **dementia** (MID) or senile **dementia** of **Alzheimer**'s type (SDAT). The results showed that in the physiological aging process, .beta.-adrenergic receptor density is reduced and 5HT transport system is enhanced. MID is associated to a loss of .beta.-adrenoceptor affinity and a slight increase in the capacity of 5HT uptake. The kinetic constants of both .beta.-adrenergic receptors in lymphocytes and 5HT uptake sites in **platelets** from SDAT patients were comparable to those observed in the adult population, being thus different from those found in the aged matched volunteers. These changes could be interpreted as resulting from compensatory mechanisms that replaced altered plasma monoamine concentrations. The apparent absence of such changes in SDAT patients could suggest the lack of these compensatory effects.

L20 ANSWER 29 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
93349397 EMBASE Document No.: 1993349397. **Platelet** of **Alzheimer** patients: Increased counts and subnormal uptake and accumulation of [14C]5-hydroxytryptamine. Inestrosa N.C.; Alarcon R.; Arriagada J.; Donoso A.; Alvarez J.. Molecular Neurobiology Unit, Dept. of Cell/Molecular Biology, Faculty of Biological Sciences, P.O. Box 114-D,Santiago, Chile. Neuroscience Letters 163/1 (8-10) 1993.
ISSN: 0304-3940. CODEN: NELED5. Pub. Country: Ireland. Language: English. Summary Language: English.

AB **Platelets** are the main source of 5-hydroxytryptamine (5-HT) and amyloid precursor protein (APP) found in plasma. We studied a possible correlation between **platelet** markers and the clinical **diagnosis** of **Alzheimer** disease (AD). Our results

indicate that in AD patients: (a) **platelets** are elevated, (b) their ability to accumulate 5-HT decreases and, (c) the kinetic parameters of 5-HT uptake are altered (decreased K(m) and V(max)), compared to non-demented healthy individuals. An aged Down syndrome patient presents even more deviant alterations. Our finding supports the idea that **platelets** may provide a systemic marker of AD, and eventually be useful for the clinical **diagnosis** of the disease.

L20 ANSWER 30 OF 47 CAPLUS COPYRIGHT 2003 ACS
1993:18858 Document No. 118:18858 Method of diagnosing or categorizing disorders from biochemical profiles. Matson, Wayne R. (ESA, Inc., USA). PCT Int. Appl. WO 9213273 A1 19920806, 42 pp. DESIGNATED STATES: W: CA, JP, RU; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1992-US375 19920116. PRIORITY: US 1991-643541 19910118; US 1991-649676 19910201.

AB A method for diagnosing disorders in living organisms is disclosed, in which fluid samples from normal and afflicted (abnormal) individuals are analyzed to generate patterns representative of mol. constituents of said samples. A data base of frequency distribution patterns of constituents of samples from organisms having known categories of disorders and controls is created, and the unknown sample anal. is compared for conformity to the frequency distribution patterns. The invention has particular applicability to diagnosing diseases, e.g. **Alzheimer**'s disease, Parkinson's disease, Huntington's disease, schizophrenia, progressive supranuclear palsy, amyotrophic lateral sclerosis, and senile **dementia**. The invention also may be advantageously employed to diagnose diseases such as tumors, carcinomas, cardiovascular abnormalities, and other disorders, or for selection of the therapy based on categories of known vs. unsuccessful outcomes. Moreover, both treatment protocols and new pharmaceuticals may be evaluated. Cerebrospinal fluid samples from patients with **Alzheimer**'s disease, Parkinson's disease, schizophrenia, Huntington's disease, and supranuclear palsy and from neurol. normal controls were analyzed by chromatog. and a 16-sensor electrochem. cell for 38 known components (e.g. adenine, cysteine, tyramine, uric acid, etc.) and for 18 well-defined unknown peaks. Linear and stepwise regression anal. were used in preliminary evaluation of the data and then cluster anal. procedures were performed. The biochem. response of controls or normal individuals was more chaotic then that of disordered individuals. Frequency distribution graphs of **Alzheimer**'s disease and controls were prepd. as well as a plot showing scoring of **Alzheimer**'s vs. control.

L20 ANSWER 31 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
93:285301 The Genuine Article (R) Number: KZ881. NONNEURAL MARKERS IN **ALZHEIMER**-DISEASE. BLASS J P (Reprint); GIBSON G E. CORNELL UNIV, COLL MED, BURKE MED RES INST, DEMENTIA RES SERV, WHITE PLAINS, NY, 10605 (Reprint). ALZHEIMER DISEASE & ASSOCIATED DISORDERS (WIN 1992) Vol. 6, No. 4, pp. 205-224. ISSN: 0893-0341. Pub. country: USA. Language: ENGLISH. *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Nonneural tissues are now widely used to search for abnormalities in genes as well as for other markers of **dementia** of the **Alzheimer** type (DAT). Studies of nonneural tissues can experimentally circumvent problems inherent in the study of autopsy brain, but to be meaningful, abnormalities identified in the periphery must be correlated with abnormalities in the brain, which is the tissue of clinical interest. Among the topics in DAT research that can be readily studied in nonneural cells (including tissue cultures) are molecular genetics, amyloid precursor protein formation and metabolism, systemic manifestations of immunological and inflammatory mechanisms, proteolysis, membranes, signal transduction, and mitochondria and metabolism. Although phenomena suggesting the possibility of cytoskeletal abnormalities in nonneural DAT cells have been described, the tau molecules involved in

paired helical filament formation are relatively brain-specific. Since the neuropathological **diagnosis** of DAT depends on recognizing a pattern of changes rather than any single abnormality, it seems unlikely that any one laboratory abnormality in peripheral tissues will correlate precisely with the clinicopathological entity of DAT. However, abnormalities found in nonneural DAT cells that correlate with the existence of similar abnormalities in the brain are likely to be informative about the disease process in the patients in whom they occur.

L20 ANSWER 32 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
92:253242 The Genuine Article (R) Number: HN397. **ALZHEIMER**-TYPE **DEMENTIA** AND VERBAL MEMORY PERFORMANCES - INFLUENCE OF SELEGILINE THERAPY. FINALI G (Reprint); PICCIRILLI M; OLIANI C; PICCININ G L. UNIV PERUGIA, NEUROL CLIN, NEUROPSICOL LAB, VIA E DAL POZZO, I-06100 PERUGIA, ITALY (Reprint). ITALIAN JOURNAL OF NEUROLOGICAL SCIENCES (MAR 1992) Vol. 13, No. 2, pp. 141-148. ISSN: 0392-0461. Pub. country: ITALY. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB In a double blind randomized crossover trial lasting 6 months selegiline, a selective MAO-B inhibitor, was tested against placebo for activity on verbal memory performances in **Alzheimer**-type **dementia** (DAT). Verbal memory was assessed with the Rey-Auditory-Verbal Learning Test at the start of treatment, at the time scheduled for crossover (90 days) and at the end of the trial (180 days). The results suggest that selegiline possesses significant activity on some memory parameters, which seems to depend on an improvement both in information processing abilities and in learning strategies at the moment of acquisition

L20 ANSWER 33 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
91:472885 The Genuine Article (R) Number: GB312. RISK-FACTORS FOR STROKE AS PREDICTORS OF **PLATELET** MEMBRANE FLUIDITY IN **ALZHEIMERS**-DISEASE. ZUBENKO G S (Reprint); BRENNER R P; TEPLY I. UNIV PITTSBURGH, WESTERN PSYCHIAT INST & CLIN, SCH MED, GERIATR HLTH SERV, 3811 OHARA ST, ROOM E-1230, PITTSBURGH, PA, 15213 (Reprint); UNIV PITTSBURGH, WESTERN PSYCHIAT INST & CLIN, SCH MED, DEPT PSYCHIAT, PITTSBURGH, PA, 15213; UNIV PITTSBURGH, WESTERN PSYCHIAT INST & CLIN, SCH MED, DEPT NEUROL, PITTSBURGH, PA, 15213. STROKE (1991) Vol. 22, No. 8, pp. 997-1003. Pub. country: USA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We have previously reported that increased **platelet** membrane fluidity identifies a subgroup of patients with **Alzheimer**'s disease who have distinct clinical features including an earlier age of symptomatic onset, a more rapidly progressive cognitive decline, and a decreased prevalence of focal electroencephalographic findings. In the current study, these patients also exhibited a decreased prevalence of risk factors for stroke compared with patients who had normal **platelet** membrane fluidity. Our findings suggest that the **platelet** membrane abnormality describes a clinical subgroup of patients with **Alzheimer**'s disease who are less likely to have coexisting cerebrovascular disease than the remaining patients who meet clinical consensus criteria for probable **Alzheimer**'s disease.

L20 ANSWER 34 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
91:596945 The Genuine Article (R) Number: GL953. **PLATELET** [H-3] PAROXETINE BINDING TO 5-HT UPTAKE SITES IN **ALZHEIMERS**-DISEASE. ANDERSSON A; ADOLFSSON R; ERIKSSON K; MARCUSSON J (Reprint). UNIV LINKOPING, DEPT GERIATR MED, S-58246 LINKOPING, SWEDEN; UMEA UNIV, DEPT GERIATR MED, S-90187 UMEA, SWEDEN; UMEA UNIV, DEPT PSYCHIAT, S-90187 UMEA, SWEDEN. NEUROBIOLOGY OF AGING (1991) Vol. 12, No. 5, pp. 531-534. Pub. country: SWEDEN. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **Platelet** serotonin (5-hydroxytryptamine, 5-HT) uptake sites were studied in a control group (n = 30) and an **Alzheimer** group (n = 40) using [H-3]paroxetine as radioligand. The maximum number of binding sites (B(max)) for control (1250 +/- 60 fmol/mg protein) was not different from the **Alzheimer** group (1280 +/- 40 fmol/mg protein). There were no differences in apparent binding affinity (K(d)): 0.046 (0.024-0.062) nM for control and 0.040 (0.027-0.061) nM for **Alzheimer**. Thus even though several previous studies have demonstrated marked atrophy of 5-HT containing neurites and 5-HT uptake sites in **Alzheimer**'s disease, these findings are not found in the periphery on **platelets**. The **platelet** 5-HT uptake site cannot be used as a peripheral marker of **Alzheimer**'s disease.

L20 ANSWER 35 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
92039093 EMBASE Document No.: 1992039093. Clinical **diagnosis** of **Alzheimer**'s disease. Burns A.. Institute of Psychiatry, De Crespigny Park, London SE5 8AF, United Kingdom. Dementia 2/4 (186-194) 1991.
ISSN: 1013-7424. CODEN: DEMNEU. Pub. Country: Switzerland. Language: English. Summary Language: English.
AB There are a number of ways in which a clinical **diagnosis** of **dementia** of the **Alzheimer** type can be made - the application of clinical criteria is the commonest but ancillary techniques such as neuroimaging, peripheral markers and neurophysiological investigations are helpful. The NINCDS/ADRDA criteria are the most consistent in correctly predicting, during life, who will have the neuropathological findings of **Alzheimer**'s disease and as such represent the gold standard for clinical **diagnosis**. **Platelet** membrane fluidity and the results of nasal biopsy may also be helpful but the specificity of abnormalities for **Alzheimer**'s disease has yet to be established. Computerised tomography and single photon emission tomography are the two most applicable and widely available neuroimaging techniques and, with single photon emission tomography, characteristic patterns of blood flow distribution are seen in **Alzheimer**'s disease. Even with the application of clinical guidelines and appropriate use of ancillary investigations, there is still diagnostic misclassification in up to 20% of cases for the refinement of clinical criteria and prospective clinico-pathological studies along with clinical criteria for other types of **dementia** are required.

L20 ANSWER 36 OF 47 MEDLINE
92024855 Document Number: 92024855. PubMed ID: 1656688. **Platelet** membrane fluidity in **Alzheimer**'s disease and multi-infarct **dementia**: a spin label study. Kaakkola S; Rosenberg P H; Alila A; Erkinjuntti T; Sulkava R; Palo J. (Department of Neurology, University of Helsinki, Finland.) ACTA NEUROLOGICA SCANDINAVICA, (1991 Jul) 84 (1) 18-21. Journal code: 0370336. ISSN: 0001-6314. Pub. country: Denmark. Language: English.
AB The membrane fluidity of **platelets** isolated from 15 patients with probable **Alzheimer**'s disease (AD), 11 patients with probable multi-infarct **dementia** (MID), and 7 neurologically healthy controls was studied by electron spin resonance (ESR) spectroscopy employing spin label techniques. Spin label I(12,3) probed the shallow site (hydrophilic region) and spin label I(5, 10) the deeper site (hydrophobic region) of the **platelet** membrane. With both probes, a significant increase in membrane fluidity was observed in patients with AD and MID, as compared to age-matched controls. However, there were no significant differences in fluidity between AD and MID patients. Our results suggest an increased **platelet** membrane fluidity in **dementias**, but the change seems not to be specific to AD.

L20 ANSWER 37 OF 47 SCISEARCH COPYRIGHT 2003 ISI (R)
91:462670 The Genuine Article (R) Number: GA689. **PLATELET** MEMBRANE FLUIDITY IN **ALZHEIMERS**-DISEASE AND MULTIINFARCT **DEMENTIA** - A SPIN LABEL STUDY. KAAKKOLA S (Reprint); ROSENBERG P H; ALILA A; ERKINJUNTTI T; SULKAVA R; PALO J. UNIV HELSINKI, DEPT NEUROL, HAARTMANINKATU 4, SF-00290 HELSINKI 29, FINLAND (Reprint); UNIV HELSINKI, DEPT ANESTHESIOL, SF-00290 HELSINKI 29, FINLAND. ACTA NEUROLOGICA SCANDINAVICA (1991) Vol. 84, No. 1, pp. 18-21. Pub. country: FINLAND. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The membrane fluidity of **platelets** isolated from 15 patients with probable **Alzheimer**'s disease (AD), 11 patients with probable multi-infarct **dementia** (MID), and 7 neurologically healthy controls was studied by electron spin resonance (ESR) spectroscopy employing spin label techniques. Spin label I(12, 3) probed the shallow site (hydrophilic region) and spin label I(5, 10) the deeper site (hydrophobic region) of the **platelet** membrane. With both probes, a significant increase in membrane fluidity was observed in patients with AD and MID, as compared to age-matched controls. However, there were no significant differences in fluidity between AD and MID patients. Our results suggest an increased **platelet** membrane fluidity in **dementias**, but the change seems not to be specific to AD.

L20 ANSWER 38 OF 47 MEDLINE
91028013 Document Number: 91028013. PubMed ID: 2171687. **Platelet** benzodiazepine binding in **Alzheimer**'s disease. Bidder M; Ratzoni G; Weizman A; Blumensohn R; Norymberg M; Tyano S; Gavish M. (Department of Pharmacology, Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel.) BIOLOGICAL PSYCHIATRY, (1990 Oct 1) 28 (7) 641-3. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

L20 ANSWER 39 OF 47 MEDLINE
89327786 Document Number: 89327786. PubMed ID: 2546986. **Alzheimer**'s **dementia** and binding to alpha 2 adrenoreceptors in **platelets**. Adunsky A; Hershkowitz M; Rabinowitz M. (Department of Geriatric Medicine and Rehabilitation, Chaim Sheba Medical Center, Tel Hashomer, Israel.) JOURNAL OF THE AMERICAN GERIATRICS SOCIETY, (1989 Aug) 37 (8) 741-4. Journal code: 7503062. ISSN: 0002-8614. Pub. country: United States. Language: English.
AB Seventy-five patients with probable **Alzheimer**'s disease were screened for binding of alpha 2 receptors (A2R) to their **platelet** membranes; the results were compared with 51 age- and sex-matched controls. Receptor binding assays were performed using [3H] Yohimbine as the radioligand. The results showed a higher binding capacity in the demented population as compared to the control group (2.18 +/- 0.15 fmol/mg protein, as compared to 1.73 +/- 0.13, P less than 0.03). This increased binding to **platelets** in the demented patients was more prominent in demented females: 34% higher binding as compared with female controls (2.06 +/- 0.5 vs 1.54 +/- 0.04). The difference between demented and normal males was less (2.34 +/- 0.05 vs 1.88 +/- 0.05). The results indicate an involvement of the A2R system, either primarily or secondarily, in the disease process. Since there is an overlap between results from the patients with **Alzheimer**'s disease and the normal subjects, A2R may serve as only a supportive marker for **Alzheimer**'s disease.

L20 ANSWER 40 OF 47 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1989:477437 Document No.: BA88:113197. A STUDY OF THE CLINICAL AND THE NEURORADIOLOGICAL FINDINGS IN MULTI-INFARCT **DEMENTIA** AND

ALZHEIMER TYPE **DEMENTIA**. ENDO R. DEP. NEUROL., NEUROL. INST., TOKYO WOMEN'S MED. COLL., TOKYO, JPN.. J TOKYO WOMEN'S MED COLL, (1989) 59 (6), 693-704. CODEN: TJIZAF. ISSN: 0040-9022. Language: Japanese.

AB In forty patients with **dementia**, a comparison of the clinical and the neuroradiological findings between 15 **Alzheimer** type **dementia** (ATD) and 21 multi-infarct **dementia** (MID) were made. MID had significantly ($p < 0.01$) higher Hachinski's Ischemic Score (HIS) (mean .+-. S.D., 9.7 .+-. 1.8) compared with ATD (3.6 .+-. 1.5). The HIS was a useful diagnostic aid in differential **diagnosis** between the two groups. MID significantly ($p < 0.01$) had cerebrovascular risk factors such as hypertension, diabetes mellitus and increase of **platelet** aggregation. It was suggested that it was important to control these factors for the prevention of MID. The morphometric analysis of the ratios of the ventricular dilatation, the cortical atrophy, and the white matter changes was performed on the CT scan and the magnetic resonance imaging. This was the first time the method of having the cortical atrophy analyzed by the ratio of the area of the sylvian sulci and the area of the whole brain had been used. It was found that the degrees of the ventricular dilatation, the cortical atrophy, and the white matter changes were more increased in MID than in ATD ($p < 0.01$ .apprx. 0.05). In ATD, there was a positive correlation between Hasegawa's **Dementia** Scale and both the ratios of the ventricular dilatation, and the cortical atrophy ($r = -0.62$, $p < 0.05$, $r = -0.63$, $p < 0.05$. respectively). Also a comparative study between MID and 9 patients with multiple infarction, without **dementia** (MI). MID had the mean infarct numbers of 6.5 .+-. 2.5, and MI had 4.1 .+-. 2.2. The white matter changes were more increased in MID than MI ($p < 0.05$). The incidence of the **dementia** was significantly higher in cases with left lenticular nucleus ($p < 0.01$) or main lesions of the white matter in the left frontal lobe ($p < 0.05$), and in cases with bilateral lenticular nucleus ($p < 0.01$), compared to cases without lesions. It was considered that the numbers of infarction, its localization, and the white matter changes may play important roles in the origin of MID.

L20 ANSWER 41 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

89165811 EMBASE Document No.: 1989165811. **Platelet** [3H]-imipramine binding is not modified in **Alzheimer**'s disease. Galzin A.-M.; Davous P.; Roudier M.; Lamour Y.; Poirier M.-F.; Langer S.Z.. Department of Biology, L.E.R.S., 75013 Paris, France. Psychiatry Research 28/3 (289-294) 1989.
ISSN: 0165-1781. CODEN: PSRSDR. Pub. Country: Ireland. Language: English. Summary Language: English.

AB **Platelet** [3H]-imipramine binding was studied in patients with **Alzheimer**'s disease and control subjects matched to the patients for age and sex. There were no differences in the binding parameters of [3H]-imipramine on **platelet** membranes from patients with **Alzheimer**'s disease, when compared with the control group. These results suggest that [3H]-imipramine binding could be a useful tool to discriminate between demented and depressive patients in elderly populations.

L20 ANSWER 42 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

89079562 EMBASE Document No.: 1989079562. Early stages of late onset **Alzheimer**'s disease. IV. Biochemical measurements suggesting metabolic derangement. Forssell L.G.; Eriksson H.. Department of Geriatric Medicine, Karolinska Institute, Huddinge University Hospital, S-141 86 Huddinge, Sweden. Acta Neurologica Scandinavica, Supplement 79/121 (67-86) 1989.
ISSN: 0065-1427. CODEN: ANSLAC. Pub. Country: Denmark. Language: English. Summary Language: English.

AB In patients with early stages of **Alzheimer**'s disease (AD), late

onset type, ordinary blood analyses such as hemoglobin, blood cell sedimentation rate, level of HbA(1c), leucocyte and **platelet** concentrations and serum levels of sodium, potassium, calcium, phosphate, creatinine, haptoglobin, aspartate amino transferase, lactate dehydrogenase, cobal-amines, folic acid, free thyroxine, and thyroid stimulating hormone showed similar average levels as in age matched, healthy controls. Erythrocyte mean corpuscular volume was lower in the late onset AD patients but was still within normal range (for middle-aged persons). Serum albumin was in the normal range (for middle-aged persons) in the late onset AD patients but significantly higher when compared to the controls. Serum levels of glucose, total protein, bilirubin, alkaline phosphatase, iron, total iron binding capacity were in the normal range (for middle-aged persons) in these AD patients. Serum levels of growth hormone and cortisol in late onset AD were significantly increased in the morning when compared to the afternoon levels. Serum estradiol was significantly reduced in male late onset AD patients when compared to controls. Cholinesterase activity in plasma and erythrocytes was significantly decreased, and the ratio of cholinesterase activity in plasma over that in erythrocytes was at the same average level as in the controls. Urinary excretions of cortisol and epinephrine were significantly increased in the late onset AD patients, when compared to the controls. Homovanillic acid and norepinephrine in urine were on similar average levels to those in the controls. So were the average serum levels of dehydroepiandrosterone, dehydroepiandrosterone sulphate, prolactin, testosterone and the serum lipids as total cholesterol, HDL-cholesterol, LDL-cholesterol and triglycerides. The frequencies of haptoglobin type 1-1 and the subtype 1 were on the same average levels in the late onset AD patients as in the controls. The implications of the results are discussed and the results are compared with those of other **dementia** studies. Some factors behind divergent results in the literature are discussed and exemplified.

L20 ANSWER 43 OF 47 MEDLINE

88339507 Document Number: 88339507. PubMed ID: 2844132. Marked reduction in the number of **platelet**-tritiated imipramine binding sites in geriatric depression. Nemeroff C B; Knight D L; Krishnan R R; Slotkin T A; Bissette G; Melville M L; Blazer D G. (Department of Psychiatry, Duke University Medical Center, Durham, NC 27710.) ARCHIVES OF GENERAL PSYCHIATRY, (1988 Oct) 45 (10) 919-23. Journal code: 0372435. ISSN: 0003-990X. Pub. country: United States. Language: English.

AB The number (Bmax) and affinity (Kd) of **platelet**-tritiated imipramine binding sites was determined in young and middle-aged controls 50 years of age and younger (n = 25), elderly normal controls over 60 years of age (n = 18), patients who fulfilled DSM-III criteria for major depression who were under 50 years of age (n = 29), patients who fulfilled DSM-III criteria for major depression who were 60 years of age and older (n = 19), and patients who fulfilled both DSM-III criteria for primary degenerative **dementia** and National Institute of Neurological and Communicative Disorders and Stroke-**Alzheimer**'s Disease and Related Disorders Association criteria for probable **Alzheimer**'s disease (n = 13). Both groups of depressed patients (under 50 and over 60 years of age) exhibited significant reductions (decreases 42%) in the number of **platelet**-tritiated imipramine binding sites with no change in affinity, when compared with their age-matched controls. There was little overlap in Bmax values between the elderly depressed patients and their controls. The patients with probable **Alzheimer**'s disease showed no alteration in **platelet**-tritiated imipramine binding. There was no statistically significant relationship between postdexamethasone plasma cortisol concentrations and tritiated imipramine binding. These results indicate that **platelet**-tritiated imipramine binding may have potential utility as a diagnostic adjunct in geriatric depression, and moreover that the reduction in the number of

platelet-tritiated imipramine binding sites is not due to hypercortisolemia.

L20 ANSWER 44 OF 47 MEDLINE

89131807 Document Number: 89131807. PubMed ID: 3223331. **Platelet** MAO-B activity and the psychopathology of Parkinson's disease, senile **dementia** and multi-infarct **dementia**. Danielczyk W; Streifler M; Konradi C; Riederer P; Moll G. (Department of Neurology, Lainz Geriatric Hospital, Vienna, Austria.) ACTA PSYCHIATRICA SCANDINAVICA, (1988 Dec) 78 (6) 730-6. Journal code: 0370364. ISSN: 0001-690X. Pub. country: Denmark. Language: English.

AB Monoamine oxidase-B (MAO-B) activity of **platelets** of an age- and sex-matched group of controls was compared with several groups of inpatients having non-familial **dementia** of **Alzheimer** type (DAT), Parkinson's disease (PD), multi-infarct **dementia** (MID), mixed types of these 3 diseases and a group of other central nervous system (CNS) organic disorders. All patients were subjected to several psychometric tests, including the Sandoz Clinical Assessment--Geriatric Scale, Hamilton Rating Scale for Depression, Mini-Mental State Examination and the Organic mental Disorder Scale (OMDS). A statistically significant enhancement of MAO-B activity could be observed in DAT patients and in PD patients, whereas the MID group showed a mean activity similar to that of the control group and the group with other organic CNS disorders. In DAT patients the degree of **dementia** in the OMDS test and the enhancement of MAO activity were positively correlated, but PD did not show such a correlation. It is concluded that the increase of MAO activity in PD and in DAT might be due to a disease-related enhanced affinity to oxygen and to such oxygen-derived radicals as superoxide or hydroxyl radicals. However, a possible drug-induced enhancement of MAO activity in PD cannot be excluded. Furthermore, the MAO-B activity values in **platelets** of individual patients or controls are not indicative of **diagnosis** or prognosis of any of these diseases and are of no disease-related specificity.

L20 ANSWER 45 OF 47 MEDLINE

88047131 Document Number: 88047131. PubMed ID: 3674232. **Platelet** MAO activity in geriatric patients with depression and **dementia**. Alexopoulos G S; Young R C; Lieberman K W; Shamoian C A. (Department of Psychiatry, New York Hospital-Cornell Medical Center, Cornell University Medical College, White Plains 10605.) AMERICAN JOURNAL OF PSYCHIATRY, (1987 Nov) 144 (11) 1480-3. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.

AB The authors studied **platelet** MAO activity in psychiatrically hospitalized geriatric patients with depression and **dementia**. **Platelet** MAO activity was higher in demented patients with and without depression and in depressed patients with reversible **dementia** than in nondemented depressed patients. The data suggest that abnormally high **platelet** MAO activity may reflect a predisposition to the development of a **dementia** syndrome.

L20 ANSWER 46 OF 47 MEDLINE

86064713 Document Number: 86064713. PubMed ID: 2999549. **Platelet** 3H-imipramine binding distinguishes depression from **Alzheimer** **dementia**. Suranyi-Cadotte B E; Gauthier S; Lafaille F; DeFlores S; Dam T V; Nair N P; Quirion R. LIFE SCIENCES, (1985 Dec 16) 37 (24) 2305-11. Journal code: 0375521. ISSN: 0024-3205. Pub. country: ENGLAND: United Kingdom. Language: English.

AB **Platelet** 3H-imipramine binding and serotonin uptake were studied simultaneously in normal subjects and in depressed, parkinsonian and **Alzheimer**'s disease patients to investigate the usefulness of these variables in the **diagnosis** of depression in the elderly.

Whereas Vmax of **platelet** serotonin uptake was significantly reduced in all patient groups compared to age matched normal subjects, the density of 3H-imipramine binding was reduced in depressed patients only. The lower Bmax values in depressed patients was independent of patient age. These data suggest that **platelet** 3H-imipramine binding may be a useful laboratory index which discriminates depression from **dementia** in the elderly.

L20 ANSWER 47 OF 47 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
83246784 EMBASE Document No.: 1983246784. Central catecholamine metabolism in vivo and the cognitive and motor deficits in Parkinson's disease. Mann J.J.; Stanley M.; Kaplan R.D.; et al.. Dep. Psychiatry, Payne Whitney Clin., Cornell Univ., Med. Coll., New York, NY 10021, United States. Journal of Neurology Neurosurgery and Psychiatry 46/10 (905-910) 1983. CODEN: JNNPAU. Pub. Country: United Kingdom. Language: English.
AB Cerebrospinal fluid levels of homovanillic acid (HVA) in unmedicated patients with Parkinson's disease were 45% of levels in control subjects. Levels of 3-methoxy-4-hydroxyphenylglycol (MHPG) and **platelet** monoamine oxidase activity (MAO) did not differ. Within the Parkinson's disease group **platelet** MAO B activity correlated with HVA (an MAO B substrate) but not MHPG (an MAO A substrate). A mild global **dementia** was found that did not correlate with the more severe motor deficit. There was a negative correlation between the motor deficit and HVA levels but not with MHPG. Cognitive functioning correlated positively with **platelet** MAO, and the ratio of HVA to MHPG levels and negatively with MHPG alone. It is postulated that dopaminergic and noradrenergic activity or the functional balance between these system may contribute to the observed cognitive dysfunction.

=> s schizophrenia
L21 185356 SCHIZOPHRENIA

=> s l21 and diagnosis
L22 35856 L21 AND DIAGNOSIS

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L24 160 DUP REMOVE L23 (74 DUPLICATES REMOVED)

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L24 ANSWER 1 OF 160 MEDLINE
2003076562 Document Number: 22475227. PubMed ID: 12587849. Effect of loxapine on peripheral dopamine-like and serotonin receptors in patients with **schizophrenia**. Singh Amarendra N; Barlas Cia; Saeedi Huma; Mishra Ram K. (Department of Psychiatry, Queen's University, Kingston, Ont.. singha@post.queensu.ca) . JOURNAL OF PSYCHIATRY AND NEUROSCIENCE, (2003 Jan) 28 (1) 39-47. Journal code: 9107859. ISSN: 1180-4882. Pub. country: Canada. Language: English.
AB OBJECTIVE: To investigate the effect of loxapine on peripheral dopamine D2-like and serotonin receptor binding and on psychotic symptoms. PATIENTS: Patients (n = 24) meeting the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, for **schizophrenia** were selected from an outpatient clinic (age range 18-70 yr). METHODS: Patients were given loxapine (dose determined by a physician) for a period of 12 weeks. There were clinic visits at before treatment began and at 6 weeks and 12 weeks of treatment. Scores on a variety of efficacy and safety scales were

recorded at each visit, and blood was drawn for receptor assays. RESULTS: Patients showed significant improvement on most psychiatric assessment scales after 6 and 12 weeks of treatment with loxapine, and both lymphocyte D2-like and 5-HTL2A **platelet** receptor binding were down-regulated after 6 and 12 weeks. The degree of receptor down-regulation was not significantly correlated with improvements in psychotic symptoms, however. CONCLUSION: Loxapine down-regulated both lymphocyte D2-like and **platelet** 5-HT2A receptors to the same extent, suggesting that both receptors are involved in the mechanism of action of loxapine in patients with **schizophrenia**.

L24 ANSWER 2 OF 160 CAPLUS COPYRIGHT 2003 ACS

2002:906554 Document No. 138:1044 G protein-coupled receptor (GPCR) microarrays for determination of GPCR gene expression profiles and uses in drug and toxin screening and diagnostics. Thirstrup, Kenneth; Madsen, Lars Siim; Jensen, Jens Bitsch; Hummel, Rene; Jensen, Bo Skaaning (Azign Bioscience A/s, Den.). PCT Int. Appl. WO 2002095065 A2 20021128, 43 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK337 20020521. PRIORITY: DK 2001-802 20010518.

AB The invention provides G protein-coupled receptor (GPCR) arrays, kits comprising GPCR arrays and methods to produce such GPCR arrays. GPCR arrays are useful in the detn. of GPCR expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved in the response of the GPCR expression. The invention relates to an GPCR array comprising a multiplicity of individual GPCR polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual GPCR polynucleotide spot comprises an GPCR polynucleotide compn. comprising a non-conserved region of an GPCR polynucleotide family member, the spots representing at least two different regions of an GPCR polynucleotide member of a family. The invention also relates to a set of primers specific for nonconserved regions of GPCR polynucleotide family members, wherein the set of primers are used in the method for the prodn. of an array according to the invention. In still a further aspect, the invention relates to a diagnostic method to det. the differences of GPCR expression profiles between two different biol. materials.

L24 ANSWER 3 OF 160 CAPLUS COPYRIGHT 2003 ACS

2002:906553 Document No. 138:1043 Transporter microarrays for the determination of transporter gene expression profiles and uses in drug and toxin screening and diagnostics. Jensen, Jens Bitsch; Madsen, Lars Siim; Gether, Ulrik; Jensen, Bo Skaaning (Azign Bioscience A/S, Den.). PCT Int. Appl. WO 2002095064 A1 20021128, 41 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK336 20020521. PRIORITY: DK 2001-803 20010518.

AB The object of the invention is to provide transporter arrays, kits comprising transporter arrays and methods to produce such transporter arrays. Transporter arrays are useful in the detn. of transporter

expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the transporter expression. The invention relates to an transporter array comprising a multiplicity of individual transporter polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual transporter polynucleotide spot comprises an transporter polynucleotide compn. comprising a non-conserved region of an transporter polynucleotide family member, the spots representing at least two different regions of a transporter polynucleotide. A set of primers specific for nonconserved regions of transporter polynucleotide family members are provided, wherein the set of primers are used in the method for the prodn. of an array according to the invention. A diagnostic method detecting the differences of transporter expression profiles between two different biol. materials is also provided.

L24 ANSWER 4 OF 160 CAPLUS COPYRIGHT 2003 ACS
2002:832908 Document No. 137:347474 Ion channel microarrays for the determination of ion channel gene expression profiles and uses in drug and toxin screening and diagnostics. Jensen, Bo Skaaning; Madsen, Lars Siim; Jensen, Jens Bitsch; Kjaer, Katrine (Neurosearch A/S, Den.). PCT Int. Appl. WO 2002086050 A2 20021031, 53 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-DK253 20020418. PRIORITY: DK 2001-635 20010420.

AB The invention provides completely novel and improved ion channel arrays, kits comprising ion channel arrays and methods to produce such ion channel arrays. Ion channel arrays are useful in the detn. of ion channel expression profiles in a certain biol. material, several biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the ion channel expression. In a first aspect the invention relates to an ion channel array comprising a multiplicity of individual ion channel polynucleotide spots stably assocd. with a surface of a solid support, wherein an individual ion channel polynucleotide spot comprises an ion channel polynucleotide compn. comprising a non-conserved region of an ion channel polynucleotide family member, the spots representing at least two different regions of an ion channel polynucleotide member of a family. In a further aspect, the invention relates to a set of primers specific for nonconserved regions of ion channel polynucleotide family members, wherein the set of primers are used in the method for the prodn. of an array according to the invention. In still a further aspect, the invention relates to a diagnostic method to det. the differences of ion channel expression profiles between two different biol. materials; said method comprises obtaining a first ion channel expression profile of a first biol. material according to the method of the present invention, obtaining a second ion channel expression profile of a second biol. material according to the method of the present invention, comparing the first and second ion channel expression profiles, and identifying any difference in the ion channel expression profile.

L24 ANSWER 5 OF 160 CAPLUS COPYRIGHT 2003 ACS
2002:736274 Document No. 137:259655 Novel peptides for the **diagnosis** of **schizophrenia**. Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 2002074793 A2 20020926, 27 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB,

GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-IL233 20020321. PRIORITY: IL 2001-142159 20010321; US 2001-PV278659 20010321.

AB Short peptides are provided, which bind to a body fluid sample obtained from a schizophrenic patient at a substantively higher level than to a body fluid sample obtained from a non-schizophrenic individual. The peptides are no more than 10 amino acids long and comprise a continuous sequence of at least 5 amino acids which consists of at least one pos. charged amino acid at one of its ends. The provided peptides, which are the putative binding sites of autoantibodies found in high levels in schizophrenic individuals, are thus useful in **diagnosis** of **schizophrenia**. Biotin-labeled peptide LVVGLCK was coated onto streptavidin-coated tubes and used to test plasma samples of schizophrenic patients and control non-schizophrenic patients in an enzyme immunoassay.

L24 ANSWER 6 OF 160 MEDLINE DUPLICATE 1

2002640358 Document Number: 22286835. PubMed ID: 12399953. State-dependent alterations in mitochondrial complex I activity in **platelets**: a potential peripheral marker for **schizophrenia** . Dror N; Klein E; Karry R; Sheinkman A; Kirsh Z; Mazor M; Tzukerman M; Ben-Shachar D. (Laboratory of Psychobiology, Department of Psychiatry, Rambam Medical Center and B Rappaport Faculty of Medicine, Technion, Haifa, Israel.) MOLECULAR PSYCHIATRY, (2002) 7 (9) 995-1001. Journal code: 9607835. ISSN: 1359-4184. Pub. country: England: United Kingdom. Language: English.

AB **Schizophrenia**, the most severe psychiatric disorder, is characterized by heterogeneity of clinical signs, often categorized into positive and negative symptoms. Among a wide array of competing biological mechanisms, altered cerebral energy metabolism and mitochondrial dysfunction have been suggested to play an important role in the pathophysiology of **schizophrenia**. In this study we investigated mitochondrial complex I in **platelets** of 113 schizophrenic patients divided into three groups (acute psychotic episode, chronic active state and residual **schizophrenia**) and 37 control subjects. Complex I was analysed at the level of enzymatic activity, mRNA and protein levels by enzyme kinetics, RT-PCR and Western blot analyses, respectively. Complex I activity in **platelets** of schizophrenic patients altered with disease state presenting high specificity and sensitivity. Thus, increased activity was associated with psychotic symptomology, while its decrease was observed in patients with residual **schizophrenia**. The relationship between the clinical state and complex I activity in **schizophrenia** was further supported by its positive correlation with the severity of patients' positive symptoms assessed by clinical ratings. In addition, similar alterations were observed at the levels of mRNA and protein of the 24- and 51-kDa iron-sulfur flavoprotein subunits of the complex. Taken together these results point to the potential of **platelet** complex I to turn into a reliable novel marker for **schizophrenia**. At present, definitive **diagnosis** depends only on descriptive behavioral and symptomatic information, therefore a peripheral measurable specific marker will contribute to **diagnosis** and monitoring of the disease.

L24 ANSWER 7 OF 160 MEDLINE DUPLICATE 2

2002639345 Document Number: 22286028. PubMed ID: 12399140. Elevated expression of integrin alpha(IIb) beta(IIIa) in drug-naive, first-episode schizophrenic patients. Walsh Marie Therese; Ryan Martina; Hillmann Andrew; Condren Rita; Kenny Dermot; Dinan Timothy; Thakore Jogin H.

(Respiratory Research Group, Smurfit Building, Beaumont Hospital, Beaumont, Ireland.) BIOLOGICAL PSYCHIATRY, (2002 Nov 1) 52 (9) 874-9. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

AB BACKGROUND: Patients with **schizophrenia** have an increased risk over the general public of developing cardiovascular illness. It is unknown if there are functional changes in **platelet** surface receptors in **schizophrenia**. We therefore analyzed the surface expression of glycoprotein (GP)Ib, the integrin receptor alpha(IIb)beta(IIIa), CD62 (P-selectin), and CD63, and investigated **platelet** function in schizophrenic patients compared with healthy volunteers. METHODS: Nineteen drug-naive, first-episode patients with a DSM IV **diagnosis** of paranoid **schizophrenia** were compared with matched healthy controls. Flow cytometry was used to assess **platelet** surface expression levels of GPIb, alpha(IIb)beta(IIIa), CD62, and CD63. Adenosine diphosphate-induced **platelet** aggregation was assayed. RESULTS: The schizophrenic patients had a significantly ($p < .0001$) increased number of 68,145 +/- 8,260.1 alpha(IIb)beta(IIIa) receptors, **platelet** compared with 56,235 +/- 8,079.4 receptors, **platelet** in healthy controls. CONCLUSIONS: Patients with **schizophrenia** have increased **platelet** expression of alpha(IIb)beta(IIIa), which may contribute to their increased risk of cardiovascular illness compared with the general population.

L24 ANSWER 8 OF 160 MEDLINE DUPLICATE 3

2002669636 Document Number: 22317504. PubMed ID: 12429357. Epinephrine- and thrombin-stimulated high-affinity GTPase activity in **platelet** membranes from patients with psychiatric disorders. Odagaki Yuji; Koyama Tsukasa. (Department of Psychiatry, Hokkaido University Graduate School of Medicine, Sapporo 060-8638, Japan.. odagaki@saitama-med.ac.jp) . PSYCHIATRY RESEARCH, (2002 Oct 10) 112 (2) 111-9. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB Although heterotrimeric guanine nucleotide-binding regulatory (G) proteins have been implicated in the pathophysiology of mental illnesses (especially mood disorders), direct evidence has been scarce. This study was designed to reveal possible abnormalities of receptor-coupled G protein function in **platelets** in patients with psychiatric disorders such as depression and **schizophrenia**. The functional status of alpha(2A)-adrenergic receptor-coupled G(i2) and thrombin receptor-coupled G proteins (G(i2)+G(q)) was determined by the increase in high-affinity GTPase activity in response to epinephrine and thrombin, respectively, in **platelet** membranes from 18 patients with mood disorders (15 unipolar and three bipolar subtype), 13 schizophrenic patients, four neurotic patients and 29 healthy control subjects. Neither alpha(2A)-adrenergic receptor-coupled G(i2) nor thrombin receptor-coupled G(q) was functionally altered in **platelets** from psychiatric patients compared with control subjects. No significant correlation was observed between these biochemical measures in **platelets** and severity of psychopathological symptoms. The functional coupling efficiency of G proteins with receptors appears intact, at least between alpha(2A)-adrenergic receptors and G(i2), and between thrombin receptors and G(q), in **platelets** from patients with psychiatric disorders.

L24 ANSWER 9 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 4

2002238131 EMBASE A conformational epitope which detects autoantibodies from schizophrenic patients. Deckmann M.; Mamillapalli R.; Schechtman L.; Shinitzky M.. M. Shinitzky, Department of Biological Chemistry, Weizmann Institute of Science, 76100 Rehovot, Israel. meir.shinitzky@weizmann.ac.il . Clinica Chimica Acta 322/1-2 (91-98) 2002.
Refs: 26.
ISSN: 0009-8981. CODEN: CCATAR.

Publisher Ident.: S 0009-8981(02)00162-6. Pub. Country: Netherlands. Language: English. Summary Language: English.

AB We previously found autoantibodies against **platelets** in schizophrenic patients. One of the **platelet** proteins that bind these antibodies is enolase. Here, we describe the isolation and sequencing of an immunoreactive peptide after enzymatic digestion of enolase. The 3-D structure of enolase indicates that, unexpectedly, this peptide is buried inside the protein. However, 3-D surface analysis leads to the identification of a conformational epitope that resembles the binding peptide and might constitute a specific binder of the autoantibodies. In a screening of antibody binding with the peptide LVVGLCK, we found in 50 serum samples of controls a mean of O.D.=0.46; s=.+-.0.21 relative enzyme immunoassay units, while in sera of 39 schizophrenic patients, we found a mean of O.D.=1.47; s=.+-.0.65; P<0.0001. Furthermore, an inverse correlation was observed between duration of **schizophrenia** and the level of the detected autoantibodies. A screening of autoantibodies in sera of various mental disorders with this peptide is currently in progress. .COPYRGT. 2002 Elsevier Science B.V.

L24 ANSWER 10 OF 160 CAPLUS COPYRIGHT 2003 ACS

2001:816459 Document No. 135:339302 Methods and compositions for enhancing cellular function through protection of tissue components. Frey, William H., II; Fawcett, John Randall; Thorne, Robert Gary; Chen, Xueqing (Healthpartners Research Foundation, USA). PCT Int. Appl. WO 2001082932 A2 20011108, 77 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US13931 20010430. PRIORITY: US 2000-PV200843 20000501; US 2000-PV230263 20000906; US 2000-PV233025 20000915.

AB Methods and compns. for enhancing cellular function through protection of tissue components, such as receptors, proteins, lipids, nucleic acids, carbohydrates, hormones, vitamins, and cofactors, by administering pyrophosphate analogs or related compds. Preferably, the invention provides a method for protecting a muscarinic acetylcholine receptor (mAChR) an/or increasing the efficacy of and agent the directly or indirectly affects a mAChR in a subject in need thereof.

L24 ANSWER 11 OF 160 CAPLUS COPYRIGHT 2003 ACS

2001:763320 Document No. 135:300485 Tyrosyl protein sulfotransferase (TPST) assay and TPST enhancers for **diagnosis** and treatment of autism and related disorders. Waring, Rosemary; Phoenix, Joanne (SHS International Ltd., UK). PCT Int. Appl. WO 2001077681 A1 20011018, 43 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-GB1569 20010405. PRIORITY: GB 2000-8326 20000406.

AB The present invention relates to a method for diagnosing or detecting a predisposition to autism and related diseases comprising assaying a bodily sample in vitro directly or indirectly for a reduced tyrosyl protein sulfotransferase (TPST) level as compared to a ref. sample, and a method of treatment for the same diseases by administering to a patient suffering

therefrom a therapeutic amt. of an enhancer of TPST.

L24 ANSWER 12 OF 160 CAPLUS COPYRIGHT 2003 ACS
2001:664344 Document No. 135:222339 **Schizophrenia** genetic **diagnosis**. Nanami, Hiroyuki; Takahashi, Hitoshi; Iritani, Shuji (Niigata University, Japan). Jpn. Kokai Tokkyo Koho JP 2001245661 A2 20010911, 38 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 2000-61775 20000307.

AB The present invention provides methods for **diagnosis** of **Schizophrenia**, evaluation of animal models, and drug screening, by measuring the expression of many genes, are disclosed. The method is based on the finding that expression of 52 proteins is altered in **Schizophrenia** in statistically significant manner. Expression of interferon-stimulated RNA formation factor ISGF-3, ras-related protein RAP-1A gene, TRK-B neurotropic factor gp145trkB receptor gene, migration-inhibiting factor-related protein MRP 8, were assessed for **diagnosis**.

L24 ANSWER 13 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2001:473263 Document No.: PREV200100473263. Proximity of genes for glucose regulation and **schizophrenia** on chromosome 10. Stone, W. S. (1); Faraone, S. V. (1); Su, J. (1); Tarbox, S. I. (1); Tsuang, M. T. (1). (1) Psychiatry, Harvard Medical School/Massachusetts Mental Health Center, Boston, MA USA. Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 307. print. Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001 ISSN: 0190-5295. Language: English. Summary Language: English.

AB Observations of impaired glucose regulation in **schizophrenia** are long-standing, although their pathological and etiological significance is uncertain. One approach to the issue is to determine whether genes that are related to glucose regulation are located in chromosomal regions related to **schizophrenia**. The analysis utilized the NIMH Genetics Initiative for **Schizophrenia** dataset, which includes a genome-wide extended family linkage analysis using GENEHUNTER PLUS. The subjects were Caucasians (n=146) who met DSM-III-R criteria for **schizophrenia**. **Schizophrenia** regions with nonparametric LOD (NPL) scores greater than 2.0 were compared with chromosomal regions that influence enzymes involved in glycolysis. In this sample, the strongest evidence for linkage occurred on chromosome 10. The two highest NPL peaks, 3.36 and 2.79, located at 10p12.33 and 10p11.1 respectively, were identified within a 21 cM segment of chromosome 10p. A 90% confidence interval was obtained for the highest NPL peak and an interval corresponding to a 1 LOD drop was obtained for the second highest NPL peak. These confidence regions cover areas of chromosome 10 that contain genes for hexokinase I (10q22), which catalyzes the conversion of glucose to glucose-6-phosphate, and for phosphofructokinase, **platelet** type (10p15.3-15.2), which catalyzes the conversion of fructose-6-phosphate to fructose 1,6 bisphosphate. Both enzymes control the rate of glycolysis. These data demonstrate that genes involved in glucose regulation are located at, or near, susceptibility regions for **schizophrenia**, and provide impetus for evaluating them as candidate genes for the disorder.

L24 ANSWER 14 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2001149339 EMBASE Serotonin-induced **platelet** intracellular calcium mobilization in various psychiatric disorders: Is it specific to bipolar disorder?. Suzuki K.; Kusumi I.; Sasaki Y.; Koyama T.. K. Suzuki, Department of Psychiatry, Hokkaido Univ. School of Medicine, North 15, West 7, Sapporo 060-8638, Japan. kasuzuki@med.hokudai.ac.jp. Journal of Affective Disorders 64/2-3 (291-296) 2001.
Refs: 44.
ISSN: 0165-0327. CODEN: JADID7.

Publisher Ident.: S 0165-0327(00)00221-4. Pub. Country: Netherlands. Language: English. Summary Language: English.

AB Background: Serotonin (5-HT)-stimulated **platelet** intracellular calcium (Ca) mobilization has been reported to be enhanced in unmedicated depressive patients compared to those of normal healthy subjects, which suggests increased 5-HT2A receptor function in these patients. It has not been ascertained whether this enhanced response is specific to some type of affective disorders among various mental disorders. Methods: We examined 5-HT-induced **platelet** intracellular Ca response in 152 unmedicated outpatients with various psychiatric disorders including bipolar disorder (BD), major depressive disorder with melancholia (DM), major depressive disorder without melancholia (DN), **schizophrenia** (SCH), panic disorder (PD), obsessive-compulsive disorder (OCD), social phobia (SP) and bulimia nervosa (BN), and 30 normal controls. Results: We observed no significant differences in basal intracellular Ca concentration among all patient subgroups and normal controls. While the 5-HT-induced Ca response was significantly and specifically higher in patients with BD than in normal controls, no significant differences were found in the Ca response to 5-HT between patients with DM, DN, SCH, PD, OCD, SP and BN, and normal controls. Limitations: The sample sizes of each group are still small. Therefore, they have to be enlarged in the continuation of the study so as to increase the power of the statistical tests. Conclusion: These results indicate the possibility that enhanced signal transduction, mediated by the 5-HT2A receptor, may be specific to bipolar disorder.

L24 ANSWER 15 OF 160 EMBASE

2001434481 EMBASE Effect of electroconvulsive therapy on hematological parameters. Chaturvedi S.; Chadda R.K.; Rusia U.; Jain N.. S. Chaturvedi, Department of Pathology, Inst. of Human Behav./All. Sciences, Delhi 110 095, India. cvsujata@id.eth.net. Psychiatry Research 104/3 (265-268) 30 Nov 2001.

Refs: 8.

ISSN: 0165-1781. CODEN: PSRSDR.

Publisher Ident.: S 0165-1781(01)00303-1. Pub. Country: Ireland. Language: English. Summary Language: English.

AB Although a complete blood count is part of the evaluation before the use of electroconvulsive therapy (ECT), there are no known hematological contraindications for the procedure. A preliminary study was done on 31 randomly selected psychiatric patients (chronic **schizophrenia**, n = 10; acute depression, n = 8; acute mania, n = 6; acute psychosis, n = 6; delusional disorder, n = 1) receiving ECT to study its hematological effects. Blood samples were drawn just before and 0, 1 and 2 h after ECT. Hemoglobin (Hb%), total and differential leukocyte count (TLC and DLC), red blood cell (RBC) count, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC) and **platelet** count were measured on a fully automated hematology analyzer (Sysmex K-1000). Significant changes were found in TLC, percentage of polymorphs and lymphocytes, and Hb%. Changes in other parameters were not statistically significant. More such studies are needed to substantiate these observations and to understand the mechanism and implication of these effects.

L24 ANSWER 16 OF 160 EMBASE DUPLICATE 5

2001170490 EMBASE The **platelet** as a peripheral marker in psychiatric illness. Plein H.; Berk M.. Prof. M. Berk, Department of Psychiatry, Faculty of Health Sciences, University of the Witwaterstarand, 7 York Road, Parktown, Johannesburg 2193, South Africa. Human Psychopharmacology 16/3 (229-236) 2001.

Refs: 67.

ISSN: 0885-6222. CODEN: HUPSEC. Pub. Country: United Kingdom. Language:

English. Summary Language: English.

AB The identification of peripheral markers of psychiatric illness is important if an improvement in the **diagnosis** and treatment of various diseases with overlapping symptomatology is desired. There are many disorders that not only have overlapping symptomatology, but also have similar biological disturbances. The functional capability of the neurons involved in the disease processes may be at the crux of the underlying pathology. The **platelet** intracellular calcium response to neurotransmitter stimulation has previously been used as a peripheral marker of psychiatric illness. This review discusses evidence in support of the extended use of the **platelet** as a peripheral marker. The use of the **platelet** intracellular calcium response to neurotransmitter stimulation as a state or trait marker in major depression, the specificity and selectivity of this response, and the possible use of the **platelet** as a peripheral marker in psychotic disorders such as **schizophrenia**, mania and psychotic depression are shown. Finally, a proposed mechanism for the association between certain psychiatric disorders and cardiovascular disease is discussed.

L24 ANSWER 17 OF 160 MEDLINE DUPLICATE 6

2001385415 Document Number: 21332922. PubMed ID: 11439238. Low **platelet** count in a 22q11 deletion syndrome subtype of **schizophrenia**. Lazier K; Chow E W; AbdelMalik P; Scutt L E; Weksberg R; Bassett A S. (Schizophrenia Research Program, Queen Street Division, Centre for Addiction and Mental Health, Ontario, M6J 1H4, Toronto, Canada.) SCHIZOPHRENIA RESEARCH, (2001 Jul 1) 50 (3) 177-80. Journal code: 8804207. ISSN: 0920-9964. Pub. country: Netherlands. Language: English.

AB Background: 22q11 Deletion Syndrome (22qDS) is a genetic syndrome associated with various physical features and **schizophrenia**. Some reports have identified thrombocytopenia (**platelet** count < 150 x 10(9)/l) in individuals with 22qDS, especially children. We investigated whether adults with 22qDS and **schizophrenia** (22qDS-SZ) have lower **platelet** counts than other patients with **schizophrenia** (SZ).Method: Complete blood counts (CBC) were recorded from medical records for 18 22qDS-SZ and 60 SZ subjects. Five CBCs per subject were randomly selected and used to calculate a within-subject mean for analyses.Results: 22qDS-SZ subjects had significantly lower mean **platelet** counts than comparison SZ subjects (142.2 x 10(9)/l versus 282.5 x 10(9)/l, $t = -11.5$, $p < 0.0001$). Ten 22qDS-SZ (55%) and no comparison subjects had thrombocytopenia.Conclusions: These results suggest that thrombocytopenia may be a common feature of 22qDS and that low **platelet** counts may comprise a readily available screening criterion to help identify this genetic syndrome among adults with **schizophrenia**.

L24 ANSWER 18 OF 160 MEDLINE DUPLICATE 7

2002116288 Document Number: 21666595. PubMed ID: 11807407. A bootstrapped commingling analysis of **platelet** monoamine oxidase activity levels corrected for cigarette smoking. Warwick Daw E; Rice J P; Anthenelli R M; Schuckit M A; Tipp J; Saccone N L; Reich T; Nurnberger J I Jr; Li T K. (Department of Epidemiology, U.T. M.D. Anderson Cancer Center, Houston, Texas, USA.) PSYCHIATRIC GENETICS, (2001 Dec) 11 (4) 177-85. Journal code: 9106748. ISSN: 0955-8829. Pub. country: England: United Kingdom. Language: English.

AB Monoamine oxidase (MAO) activity levels have been suggested as a possible biological marker for alcohol dependence and abuse, as well as for **schizophrenia** and other psychiatric conditions. Using **platelet** MAO activities in the Collaborative Study on the Genetics of Alcoholism data set, we applied bootstrapping methods as a novel way to test for admixture in families. This bootstrapping involved resampling in

family units and hypothesis testing of the resampled datasets for commingling in the distribution of MAO activity levels. Prior to commingling analysis, we used linear models to find covariates of greatest effect on MAO activity levels. While an alcoholism **diagnosis** was significant in men (n = 1151, P < 0.0001) and women (n = 1254, P = 0.0003), the effect lost significance after controlling for cigarette smoking, indicating alcoholism and smoking behavior to be highly confounded. When smoking histories were compared, former smokers had levels (mean = 7.1) closer to those who never smoked (mean = 7.0) than to current smokers (mean = 5.4). Furthermore, current daily smoking and time since smoking cessation were significantly related to MAO levels, indicating smoking probably has a direct effect on MAO levels, rather than the reverse. These results suggest that studies using MAO levels as a biological marker should consider smoking as an important covariate. Finally, admixture was found in MAO levels controlled for smoking and sex, possibly indicating a major genetic locus; this confirms previous evidence for admixture.

L24 ANSWER 19 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

2001385229 EMBASE Nitrite content and antioxidant enzyme levels in the blood of **schizophrenia** patients. Srivastava N.; Barthwal M.K.; Dalal P.K.; Agarwal A.K.; Nag D.; Srimal R.C.; Seth P.K.; Dikshit M.. M. Dikshit, Pharmacology Division, Central Drug Research Institute, Lucknow 226001, India. madhudikshit@yahoo.com. Psychopharmacology 158/2 (140-145) 2001.

Refs: 38.

ISSN: 0033-3158. CODEN: PSCHDL. Pub. Country: Germany. Language: English. Summary Language: English.

AB Rationale: Recent studies have suggested augmentation in the inflammatory response as well as involvement of nitric oxide (NO) in mood disorders. Polymorphonuclear leukocytes (PMN), NO and free radicals have been associated with inflammatory response; however, the status of NO in the PMN has not been investigated so far in **schizophrenia** patients. Objectives: The present study was undertaken to investigate levels of nitrite (a metabolite of NO), malonaldehyde (MDA, lipid peroxidation product) and antioxidant enzymes such as superoxide dismutase (SOD), catalase and glutathione peroxidase (Gpx) in the PMN of **schizophrenia** patients. Methods: Patients with **schizophrenia** (n=62) were diagnosed according to DSM-IV and were free of anti-psychotic medications/ECT for at least 3 months. Mean age of the patients was 29.06.+-.1.17 years, with a male to female ratio of 4:1, and mean duration of illness was 3.7.+-.0.6 years. The control group consisted of 82 healthy subjects with a mean age of 37.0.+-.1.26 and a male to female ratio of 5:1. PMN were isolated from the blood. Nitrite, MDA and antioxidant enzymes were estimated by standard biochemical techniques in the PMN of normal healthy controls and **schizophrenia** patients. **Platelet** and plasma nitrite levels were also estimated in controls and **schizophrenia** patients. Results: Nitrite content in the PMN was reduced to 68%, while plasma and **platelet** nitrite content in **schizophrenia** patients was not significantly changed in comparison to controls. Malonaldehyde (MDA) content in PMN was significantly augmented in **schizophrenia** patients but activity of SOD, catalase and Gpx remain unaltered. Conclusion: Results obtained indicate a significant decrease in NO synthesis and an increase in MDA in the PMN of **schizophrenia** patients, while antioxidant enzyme activities were not altered in the PMN of **schizophrenia** patients. This suggests that the decrease in PMN NO synthesis by PMN might lead to oxidative stress in **schizophrenia** patients.

L24 ANSWER 20 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

2001157853 EMBASE Stereotyped behaviors in chronic **schizophrenia** in a Japanese Mental Hospital. Kaneda Y.; Fujii A.; Ohmori T.. Y. Kaneda,

Department of Neuropsychiatry, School of Medicine, Univ. of Tokushima, Tokushima-shi, Tokushima 770-8503, Japan. International Medical Journal 8/1 (35-39) 2001.
Refs: 41.
ISSN: 1341-2051. CODEN: IMJOFS. Pub. Country: Japan. Language: English. Summary Language: English.

AB Objective: The authors investigated the relationships between stereotyped behaviors and plasma homovanillic acid (HVA), **platelet** serotonin (5-HT) levels, and psychopathology in chronically medicated schizophrenic inpatients. Design: The subjects were 30 inpatients who were diagnosed according to the DSM-IV criteria for **schizophrenia**. Each patient gave informed consent for the research involved in this study. Stereotyped behaviors and other psychiatric symptoms were assessed using the Japanese-language version of the Elgin Behavior Rating Scale (JEBRS), and BPRS. Results: (1) Smoking obtained the highest mean rating, bizarre grooming and hypersexuality the lowest. Five out of 30 (16.7%) patients had at least one severe or two moderate stereotyped behaviors. (2) The total score for stereotyped behaviors in the JEBRS was positively correlated with positive (POS) and negative symptoms (NES). Among five sub-groups of stereotyped behaviors, 'oral consumption' and 'movement' behaviors were positively correlated with POS, but neither with NES, plasma HVA, nor **platelet** 5-HT. In contrast, neither 'Kluver-Bucy' behaviors, 'bizarre use of objects' nor 'pica' was correlated with POS, NES, HVA, or 5-HT. Conclusion: Our results showed that (1) stereotyped behaviors might be related closely to the POS and weakly to NES, but (2) plasma HVA and **platelet** 5-HT might not reflect severity of stereotyped behaviors for a group of patients in a clinical setting where a wide range of antipsychotic doses was being prescribed.

L24 ANSWER 21 OF 160 SCISEARCH COPYRIGHT 2003 ISI (R)
2001:409762 The Genuine Article (R) Number: 432NP. Depression, anxiety, and the cardiovascular system: The cardiologist's perspective. Sheps D S (Reprint); Sheffield D. Univ Florida, Sch Med, Div Cardiovasc Med, POB 100277, Gainesville, FL 32610 USA (Reprint); Univ Florida, Sch Med, Div Cardiovasc Med, Gainesville, FL 32610 USA. JOURNAL OF CLINICAL PSYCHIATRY (MAY 2001) Vol. 62, Supp. [8], pp. 12-18. Publisher: PHYSICIANS POSTGRADUATE PRESS. P O BOX 240008, MEMPHIS, TN 38124 USA. ISSN: 0160-6689 . Pub. country: USA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Up to one fifth of patients with cardiovascular disease, including those who have experienced a myocardial infarction, may have concomitant major depression. Studies have suggested that the relative risk of major depression with cardiovascular disease ranges from 1.5 to 4.5. Further information is required to establish a dose-response relationship between depression and coronary artery disease (CAD); however, such a relationship has been shown between anxiety and CAD. Development of a conceptual model of the pathophysiologic actions of stress in CAD will assist in the understanding of this relationship. In patients with angiographic evidence of CAD, the presence of major depressive disorder was the best single predictor of cardiac events during the 12 months following **diagnosis**. Significantly, 6-month cumulative mortality following **diagnosis** of myocardial infarction has been shown to be higher in depressed patients than in nondepressed patients. A decrease in heart rate variability may mediate the deleterious effect of depression on post-myocardial infarction prognosis. Other factors such as mental stress and altered **platelet** function may also predispose depressed patients to a heightened risk of cardiac events. With an increased understanding of the relationship be tween depression and heightened risk of cardiovascular mortality, it is necessary to assess current overall treatment for cardiac patients.

L24 ANSWER 22 OF 160 CAPLUS COPYRIGHT 2003 ACS
2000:241564 Document No. 132:288780 Methods of identifying inverse agonists of the serotonin 2a receptor, therapeutic and diagnostic methods, and test kit. Weiner, David; Brann, Mark R. (Acadia Pharmaceuticals Inc., USA). PCT Int. Appl. WO 2000020636 A1 20000413, 42 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-US21439 19991007. PRIORITY: US 1998-103317 19981007; US 1999-413626 19991006.

AB A method for identifying compds. which act as inverse agonists of the 5-HT2A receptor comprises contacting a constitutively active 5-HT2A receptor with at least one test compd. and detg. any decrease in the level of basal activity of the receptor. The inverse agonists may be used in the treatment of **schizophrenia** and related psychoses.

L24 ANSWER 23 OF 160 CAPLUS COPYRIGHT 2003 ACS
2000:227858 Document No. 132:260666 Identifying agents that alter mitochondrial permeability transition pores and cell death for diagnostic and therapeutic use. Dykens, James A.; Miller, Scott W.; Ghosh, Soumitra S.; Davis, Robert E. (Mitokor, USA). PCT Int. Appl. WO 2000019200 A1 20000406, 88 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-US22261 19990924. PRIORITY: US 1998-161172 19980925.

AB Methods are provided for identifying agents that affect mitochondrial functions and cell death. Such agents are useful for treating diseases assocd. with mitochondrial dysfunction and in methods of identifying a risk or presence of such diseases. In particular, the invention relates to the loss of mitochondrial membrane potential (.DELTA..PSI.m) during mitochondrial permeability transition (MPT) and further provides a measurable rate loss function, changes in which are useful e.g. for detecting agents that affect one or more mitochondrial functions, for detecting mitochondrial diseases, and for studying mol. components of mitochondria that regulate MPT.

L24 ANSWER 24 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2000307085 EMBASE Prospective evaluation of circulatory levels of catecholamines and serotonin in neuroleptic malignant syndrome. Spivak B.; Maline D.I.; Vered Y.; Kozyrev V.N.; Mester R.; Neduva S.A.; Ravilov R.S.; Graff E.; Weizman A.. Dr. B. Spivak, Ness-Ziona Mental Health Center, POB 1, Ness-Ziona 74100, Israel. Acta Psychiatrica Scandinavica 102/3 (226-230) 2000.
Refs: 23.
ISSN: 0001-690X. CODEN: APYSA. Pub. Country: Denmark. Language: English. Summary Language: English.

AB Objective: Neuroleptic malignant syndrome (NMS) may be associated with a dysregulation of the catecholaminergic and serotonergic systems. The objective of the present study was to evaluate prospectively the circulatory levels of serotonin (5-HT), epinephrine (E) and dopa in patients suffering from NMS. Method: **Platelet**-poor plasma (PPP) levels of serotonin, epinephrine and dopa in eight NMS patients were measured twice: In the acute state and in the state of remission. Results:

PPP dopa concentration was significantly lower in acute NMS state compared to the remission state (P=0.023). In contrast, PPP E level was significantly higher (P = 0.019) in the acute NMS state and PPP 5-HT concentrations in the acute state tended to be higher than those at remission (P= 0.078). 5-HT/dopa ratio was significantly higher in the acute NMS (P=0.015). Conclusion: These results may reflect reduction in dopaminergic function and increase in adrenergic and serotonergic activity in the acute NMS state.

L24 ANSWER 25 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2000343789 EMBASE [Infantile autism and **schizophrenia**: Comparative ultrastructural study in the peripheral blood]. AUTISMO INFANTIL Y ESQUIZOFRENIA: ESTUDIO ULTRAESTRUCTURAL COMPARATIVO DE LA SANGRE PERIFERICA. Castillo S.M.; Martinez H.; Bestard A.; Gonzalez E.; Perez A.; Ancheta O.; Ramos M.E.; Perez N.; Valdes T.; Rodriguez S.; Dominguez C.; Melendez C.; Subiaul Z.. S.M. Castillo, Hospital Psiquiatrico de La Habana, Havana, Cuba. Revista del Hospital Psiquiatrico de la Habana 40/3 (214-219) 2000.
Refs: 36.
ISSN: 0138-7103. CODEN: HPSRAJ. Pub. Country: Cuba. Language: Spanish. Summary Language: English; Spanish.
AB A comparative ultrastructural study is done between the findings obtained in the peripheral blood from schizophrenic patients and those obtained in a similar study in the peripheral blood of an infantile autism. The vacuolization of **platelets** was a common element but with different characteristics. Intranuclear inclusions and bacterias related to virus like particles were observed in both pathologies. The absence of similar studies in the world medical literature enhances the importance of the results obtained.

L24 ANSWER 26 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2000148160 EMBASE Paroxetine binding in aggressive schizophrenic patients. Modai I.; Gibel A.; Rauchverger B.; Ritsner M.; Klein E.; Ben-Shachar D.. I. Modai, Sha'ar Menashe Mental Hlth. Ctr., Mobile Post Hefer 38814, Israel. shrmodai@matat.health.gov.il. Psychiatry Research 94/1 (77-81) 24 Apr 2000.
Refs: 25.
ISSN: 0165-1781. CODEN: PSRSDR.
Publisher Ident.: S 0165-1781(00)00116-5. Pub. Country: Ireland. Language: English. Summary Language: English.
AB Decreased central serotonergic activity has been associated with aggressive behavior in humans and animals. Whether or not this phenomenon is related to current aggression or to aggressive tendency is debatable. [3H]paroxetine binding in blood **platelets** represents the activity of serotonin peripheral binding sites. We investigated a possible association between [3H]paroxetine binding in blood **platelets** and current aggression or homicidal history in schizophrenic patients. Blood **platelets** of 11 aggressive schizophrenic patients were assayed for [3H]paroxetine binding in blood **platelets** and compared to findings in 15 non-aggressive schizophrenic patients, 15 presently non-aggressive schizophrenic patients with homicidal history, and 15 healthy volunteers. Clinical evaluation was performed using the Positive and Negative Syndrome Scale, the Hamilton Rating Scale for Depression and the Clinical Global Impression scale. B(max) of [3H]paroxetine binding in blood **platelets** of currently aggressive schizophrenic patients was significantly higher than that in **platelets** of non-aggressive schizophrenic patients, presently non-aggressive patients with homicidal history and healthy volunteers. No difference was found between the last three study groups. No significant correlation was found between scores of all rating scales and the investigated biochemical parameters. An association was found between current aggression among schizophrenic patients and high B(max) values of

[3H]paroxetine binding in blood **platelets**. This association is probably related to present state of aggression rather than to tendency towards aggression. Copyright (C) 2000 Elsevier Science Ireland Ltd.

L24 ANSWER 27 OF 160 MEDLINE DUPLICATE 8
2000256736 Document Number: 20256736. PubMed ID: 10798826. Neuroleptic malignant syndrome and severe thrombocytopenia: case report and literature review. Ghani S O; Ahmed W; Marco L A. (Department of Psychiatry, University of South Alabama, Mobile, USA.) ANNALS OF CLINICAL PSYCHIATRY, (2000 Mar) 12 (1) 51-4. Ref: 17. Journal code: 8911021. ISSN: 1040-1237. Pub. country: United States. Language: English.

AB We report an unusual case of thrombocytopenia associated with neuroleptic malignant syndrome (NMS). A 31-year-old Black male with a history of hypertension, partial seizures, and **schizophrenia** developed acute rigidity closely followed by severe hyperpyrexia (temperature 102 degree F), tachypnea, and tachycardia. His home medications at the time of presentation included propanolol 10 mg tid, haloperidol 10 mg bid, sodium valproate 500 mg bid, benztropine 1 mg bid, and haloperidol decanoate 100 mg i.m. every 3 weeks, from another psychiatric facility. Despite vigorous therapy for the hyperthermia, he rapidly developed significant hypoxia requiring mechanical ventilation. A **diagnosis** of neuroleptic malignant syndrome was made and the patient continued to receive aggressive supportive care. On hospital day 2 his **platelet** count dropped to 47,000/microl and bottomed out at 36,000/microl by day 3 with other blood cell counts remaining within normal limits. Over the next few days he showed rapid clinical improvement with normalization of his blood chemistries and he was discharged home after 5 days of hospitalization in good condition.

L24 ANSWER 28 OF 160 MEDLINE DUPLICATE 9
2000308514 Document Number: 20308514. PubMed ID: 10849968. [Specific aspects of thrombocyte system of serotonin in patients with different manifestations of schizoaffective psychosis]. Osobennosti serotoninovoi sistemy trombotsitov bol'nykh s razlichnymi klinicheskimi proiavleniiami shizofaffektivnogo psikhoza. Brusov O S; Dikaia V I; Zlobina G P; Faktor M I; Pavlova O A; Bologov P V; Korenev A N. ZHURNAL NEVROLOGII I PSIKHIATRII IMENI S.S. KORSAKOVA, (2000) 100 (5) 50-4. Journal code: 9712194. Pub. country: RUSSIA: Russian Federation. Language: Russian.

AB 45 women with different manifestations of schizoaffective psychosis (SAP) were examined. The **diagnosis** corresponded to ICD-10 (F25). According to the classification elaborated in Mental Health Research Centre of Russian Academy of Medical Sciences, groups of patients were identified with different variants of the psychoses course: a nuclear SAP type; a borderline SAP variation with phasic-recurrent course; SAP with progredient variation (schizoaffective variation of **schizophrenia**). The patients were examined both during the attack and remission. A rate of serotonine uptake (Vmax) in blood **platelets**, a specific imipramine binding (Bmax) and the level of serotonin in blood **platelets** were evaluated. It was found that dynamics of both Vmax and the level of serotonin in different SAP types were different, that was related to clinical and biological SAP heterogeneity. A tendency to decreasing of serotonin system functional activity was found in progredient SAP variations, especially during the remission, which was of low quality in these cases. On the contrary, in the borderline variations the indices of the decreased function of serotonin system corresponded well to those of acute psychosis. In nuclear type--a type with the most favourable course of psychosis--any significant changes weren't revealed as compared with the normal parameters.

L24 ANSWER 29 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2000:299632 Document No.: PREV200000299632. **Diagnosis** of the susceptibility of contracting **schizophrenia**. Shinitzky, Meir

(1). (1) Kfar Shmaryahu Israel. ASSIGNEE: Yeda Research and Development Co. Ltd., Rehovot, Israel. Patent Info.: US 6008001 December 28, 1999. Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 28, 1999) Vol. 1229, No. 4, pp. No pagination. e-file. ISSN: 0098-1133. Language: English.

AB There is described an assay for the **diagnosis** of a mental disorder in an individual. A blood sample, a **platelet**-containing fraction thereof, or a fraction containing **platelet**-associated antibodies (PAA) shed from the **platelets** is withdrawn from the individual to be diagnosed. The withdrawn sample is contacted with an anti-human immunoglobulin antibody lacking the Fc domain (Fc-less anti-hIg antibody) and the degree of binding thereof to the PAA is determined. A degree of binding above that found in normal individuals indicates that diagnosed individual has a high likelihood of having a mental disorder.

L24 ANSWER 30 OF 160 CAPLUS COPYRIGHT 2003 ACS

1999:659490 Document No. 131:270940 Assay for the **diagnosis** of **schizophrenia** based on a new peptide. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9951725 A2 19991014, 37 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-IL190 19990330. PRIORITY: IL 1998-123925 19980402.

AB The invention concerns peptides which bind antibodies that are found in elevated levels in body fluids of schizophrenic patients and are found at a lower level or not found at all in body fluids of non-schizophrenic individuals. Using a computerized program, the antigenic epitope of the peptides of the invention is predicted as having a core of hydrophobic amino acids which is surrounded by pos. charged amino acids. The peptides of the invention are useful in the **diagnosis** of **schizophrenia** in an individual.

L24 ANSWER 31 OF 160 CAPLUS COPYRIGHT 2003 ACS

1999:390463 Document No. 131:16115 Skin test for **schizophrenia**. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9930163 A1 19990617, 21 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-IL592 19981207. PRIORITY: IL 1997-122490 19971207.

AB A diagnostic method for assaying **schizophrenia** in a subject is provided wherein a prepn. comprising **platelet** derived proteins or fractions thereof having a pI above about 6.5 is injected into a subject and the occurrence of delayed type hypersensitivity (DTH) reaction at the site of the injection is detd. A pos. DTH reaction indicates that the tested subject has a high likelihood of being schizophrenic. The protein prepn. used in the diagnostic method is also provided as well as a method for its prepn. and a kit for use in the **diagnosis** of **schizophrenia** using the above method.

L24 ANSWER 32 OF 160 SCISEARCH COPYRIGHT 2003 ISI (R)

1999:415883 The Genuine Article (R) Number: 199VK. **Platelet** serotonin, plasma cortisol, and dexamethasone suppression test in

schizophrenic patients. MuckSeler D (Reprint); Pivac N; Jakovljevic M; Brzovic Z. RUDJER BOSKOVIC INST, LAB MOL NEUROPHARMACOL, POB 1016, HR-10001 ZAGREB, CROATIA (Reprint); UNIV PSYCHIAT CLIN, CLIN HOSP CTR, ZAGREB, CROATIA; CROATIAN INST BRAIN RES, ZAGREB, CROATIA. BIOLOGICAL PSYCHIATRY (1 JUN 1999) Vol. 45, No. 11, pp. 1433-1439. Publisher: ELSEVIER SCIENCE INC. 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010. ISSN: 0006-3223. Pub. country: CROATIA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background: Serotonin (5-HT) regulates hypothalamic-pituitary-adrenal (HPA) axis activity. Abnormal response to the dexamethasone suppression test (DST) and altered **platelet** 5-HT concentration have been shown in some schizophrenic patients.

Methods: **Platelet** 5-HT and plasma cortisol concentrations were determined simultaneously in 86 male schizophrenic patients before and after DST. Basal plasma cortisol and **platelet** 5-HT levels were also determined in 69 healthy male persons.

Results: Schizophrenic patients had higher plasma cortisol and **platelet** 5-HT concentrations than healthy persons. An abnormal escape from dexamethasone suppression was observed in 50% of patients. In these patients pre-dexamethasone cortisol and **platelet** 5-HT concentrations were higher than in patients with normal DST.

Conclusions: This study demonstrates that schizophrenic patients have the HPA axis dysregulation that could be connected with a disturbance in the 5-HT system. (C) 1999 Society of Biological Psychiatry.

L24 ANSWER 33 OF 160 MEDLINE DUPLICATE 10
2000129198 Document Number: 20129198. PubMed ID: 10667736. At issue: **schizophrenia** and rheumatoid arthritis: the negative association revisited. Oken R J; Schulzer M. (New York State Institute for Basic Research in Developmental Disabilities, Staten Island, USA.) SCHIZOPHRENIA BULLETIN, (1999) 25 (4) 625-38. Journal code: 0236760. ISSN: 0586-7614. Pub. country: United States. Language: English.

AB A strong negative association between **schizophrenia** and rheumatoid arthritis (RA), implying low comorbidity, has been found in 12 of 14 previous studies, which we review. To this literature we add two recently acquired data sets encompassing 28,953 **schizophrenia** patients, only 31 of whom had comorbid RA. Integrating our new data into those of the previous nine studies, which stratified their populations according to psychiatric **diagnosis**, we obtain a median frequency of RA in **schizophrenia** populations of 0.09 percent and a mean frequency of 0.66 percent, well below the expected range of 1 percent. These data robustly support prior studies. We also present a meta-analysis evaluating the association between the two diseases by integrating information derived from nine data sets, each furnishing an estimate of the relative risk of RA in **schizophrenia** patients versus that in other psychiatric patients. We find that the estimated rate of RA among **schizophrenia** patients is only 29 percent of the corresponding rate in other psychiatric patients. Further, the relative risk of RA in **schizophrenia** patients versus that in the general population is even less than 29 percent and could be as low as one-third of this value. We present a new hypothesis involving the **platelet** activating factor system in an effort to account for this negative association and review the suggestions of other investigators toward this end. Finally, we consider the glutamatergic system dysfunction hypothesis of **schizophrenia** and suggest a possible common pharmacological approach that may ameliorate some of the symptomatology of both **schizophrenia** and RA.

L24 ANSWER 34 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2000342767 EMBASE [**Schizophrenia**: Cytopathological diagnose assessment]. ESQUIZOFRENIA: ESCALA DE EVALUACION CITOPATOLOGICA DIAGNOSTICA. Mesa Castillo S.. S. Mesa Castillo, Grado en Neurologia,

Hospital Psiquiatrico de La Habana, La Habana, Cuba. Revista del Hospital Psiquiatrico de la Habana 40/2 (125-128) 1999.
Refs: 9.
ISSN: 0138-7103. CODEN: HPSRAJ. Pub. Country: Cuba. Language: Spanish. Summary Language: English; Spanish.

AB A biological test is performed from blood samples of 30 schizophrenic patients and 20 controls. The ultraestructural alterations found on **platelets** and the presence of viral like particles are useful elements for **diagnosis**, prognosis, research, epidemiology and forensic medicine that let us distinguish between a schizophrenic from a normal and can constitute itself a new element favoring the viral hypothesis of this disease. A **diagnosis** evaluation scale is applied grouping all the alterations found, and relates the highest scores to the worst condition of the disease, giving us a range above that which is considered pathological.

L24 ANSWER 35 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

1999115337 EMBASE Supersensitive **platelet** glutamate receptors as a possible peripheral marker in **schizophrenia**. Berk M.; Plein H.; Csizmadia T.. M. Berk, Department of Psychiatry, University of the Witwatersrand, Medical School, 7 York Road, Parktown 2193, South Africa. International Clinical Psychopharmacology 14/2 (119-122) 1999.
Refs: 17.
ISSN: 0268-1315. CODEN: ICLPE4. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB Hypoglutamatergic function is implicated in the pathogenesis of **schizophrenia**. The aim of this study was to examine the **platelet** intracellular calcium response to glutamate using spectroflourometry in 15 schizophrenic patients and 15 matched control individuals as an index of **platelet** glutamate receptor sensitivity. Patients with **schizophrenia** had significantly lower baseline intracellular calcium levels than matched control individuals ($P = 0.03$). The percentage response of the schizophrenic individuals to glutamate stimulation was significantly greater than control individuals ($P < 0.001$). These data suggest that **platelet** glutamate receptors may be supersensitive in **schizophrenia**. Furthermore, the **platelet** may be a possible peripheral marker of glutamate function in **schizophrenia**.

L24 ANSWER 36 OF 160 MEDLINE

1999177784 Document Number: 99177784. PubMed ID: 10078058. [Brain isoforms of creatine kinase in health and mental diseases: Alzheimer's disease and **schizophrenia**]. Mozgovaia izoforma kreatinfosfokinazy v norme i pri psikhicheskikh zabolevaniiakh (bolezn' Al'tsgemera, shizofreniia). Burbaeva G Sh; Savushkina O K; Dmitriev A D. VESTNIK ROSSIISKOI AKADEMII MEDITSINSKIKH NAUK, (1999) (1) 20-4. Ref: 39. Journal code: 9215641. ISSN: 0869-6047. Pub. country: RUSSIA: Russian Federation. Language: Russian.

AB The paper analyzes the authors' own findings and the data available in the literature on the intensity, site, and possible causes of impairment of the creatine-creatine phosphate system of brain energy metabolism in mental diseases, such as Alzheimer's disease (AD) and **schizophrenia**. Examining the level of cytosolic BB creatine kinase in postmortem AD and schizophrenic's brain structures showed a significant decrease in BB creatine kinase as compared with the similar control brain structures. There was the maximum decline in AD cases. It was considerable as compared with both the control and schizophrenic groups ($p < 0.01$). The decrement was revealed by various techniques, including the determination of activity, immunological responsiveness and the analysis of two-dimensional protein maps. Immunocytochemical investigation indicated a decrease in responses to BB creatine kinase, mainly in astrocytes. The reduction in cytosolic BB creatine kinase

levels is not a result of age, postmortem delay, or psychotic therapy. The causes of lower BB creatine kinase levels in the cell cytosol of the postmortem brain in mental pathology are discussed. The decrement in cytosolic BB creatine kinase in AD and **schizophrenia** occurs not only in the brain, but also in the peripheral tissues which contain BB creatine kinase. In all cases, it is greater in AD than in **schizophrenia**. Using immunosorbents with monoclonal antibodies to M-creatine kinase and to B-creatine kinase subunits makes it possible detect BB-creatine kinase in the extracts of human peripheral lymphocytes and **platelets**. A study of whether there is a relationship between the clinical data of mental patients and the level of BB creatine kinase in their blood elements is assumed to be useful in evaluating BB creatine kinase as a prognostic/diagnostic marker of mental diseases.

L24 ANSWER 37 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
1999299142 EMBASE What can the investigation of phosphoinositide signaling system In **platelets** of schizophrenic patients tell us?.
Strunecka A.; Ripova D.. A. Strunecka, Dept. Physiology Developmental Biol., Faculty of Sciences, Charles University, Vinicna 7, 128 00 Prague 2, Czech Republic. astrun@cesnet.cz. Prostaglandins Leukotrienes and Essential Fatty Acids 61/1 (1-5) 1999.
Refs: 47.
ISSN: 0952-3278. CODEN: PLEAEU. Pub. Country: United Kingdom. Language: English. Summary Language: English.
AB Disturbances in the regulation of the phosphoinositide signaling system have been proposed as a possible biological marker of **schizophrenia**. This review considers the laboratory investigations of phosphoinositide metabolism in **platelets** of schizophrenic patients. We suggest that alterations in the inositol phosphate level and a disturbance of calcium homeostasis may be common denominators for the multiple factors implicated in the pathogenesis of **schizophrenia** . In addition, these abnormalities may account for the diverse clinical and biochemical manifestations of **schizophrenia**.

L24 ANSWER 38 OF 160 CAPLUS COPYRIGHT 2003 ACS
1998:793038 Document No. 130:20605 New BPC peptide salts with organo-protective activity, the process for their preparation and their use in therapy. Sikiric, Predrag; Petek, Marijan; Seiwerth, Sven; Turkovic, Branko; Grabarevic, Zeljko; Rotkvic, Ivo; Mise, Stjepan; Duvnjak, Marko; Udovicic, Ivan (Croatia). PCT Int. Appl. WO 9852973 A1 19981126, 81 pp. DESIGNATED STATES: W: AU, BA, BG, BR, CA, CN, CU, CZ, EE, GE, HU, ID, IL, JP, KR, MX, NO, NZ, PL, SK, TR, UA, US, UZ, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1998-EP2953 19980520. PRIORITY: EP 1997-108384 19970523.
AB The present invention discloses new pharmaceutical compns. useful for the treatment of various human and animal diseases. These pharmaceutical compns. contain one or more salts of BPC (Body Protection Compd.) peptide comprising 8-15 amino acid residues or analogs thereof. The cations of the salts are derived from inorg. or org. bases. Thus, the prepn. of monosodium salt of BPC157 (NaBPC157) and its formulation into capsules contg. trehalose and soln. contg. glycerol are presented. Antiulcer, vascular endothelium protective, anti-angiogenesis, anti-inflammatory, free radical scavenging, cytoprotective and organ protective, cardioprotectant, antiarrhythmic, anti-parkinsonian, antihypertensive, antitumor, analgesic, anti-ischemic, etc. activities of NaBPC157 are demonstrated in animal models.

L24 ANSWER 39 OF 160 CAPLUS COPYRIGHT 2003 ACS
1998:568669 Document No. 129:185107 Cloning and cDNA sequence of a human G-protein coupled receptor (HTADX50) and its diagnostic and therapeutic uses. Bergsma, Derk J.; Ellis, Catherine E. (Smithkline Beecham Corp.,

USA). Eur. Pat. Appl. EP 859053 A1 19980819, 24 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO. (English). CODEN: EPXXDW. APPLICATION: EP 1997-309253 19971118. PRIORITY: US 1997-788750 19970124.

AB HTADX50 polypeptides and polynucleotides and methods for producing such polypeptides by recombinant techniques are disclosed. The cDNA encoding human HTADX50 was first identified from a human activated T-cell cDNA library, and contains an open reading frame encoding a protein of 330 amino acids with a deduced mol. wt. of 37.1 kDa. HTADX50 has about 28% identity in 293 amino acid residues with the thrombin receptor, and is also homologous to **platelet**-activating factor receptor and the ATP receptor; the cDNA has about 60.1% identity in 972 nucleotide residues with human B-cell receptor cDNA and is also homologous to interleukin-8 receptor cDNA. Also disclosed are methods for utilizing HTADX50 polypeptides and polynucleotides in the design of protocols for the treatment of infections such as bacterial, fungal, protozoan and viral infections, particularly infections caused by HIV-1 or HIV-2; pain; cancers; anorexia; bulimia; asthma; Parkinson's disease; acute heart failure; hypotension; hypertension; urinary retention; osteoporosis; angina pectoris; myocardial infarction; ulcers; asthma; allergies; benign prostatic hypertrophy; and psychotic and neurol. disorders, including anxiety, **schizophrenia**, manic depression, delirium, dementia, severe mental retardation and dyskinesias, such as Huntington's disease or Gilles dela Tourett's syndrome, among others and diagnostic assays for such conditions.

L24 ANSWER 40 OF 160 MEDLINE

1999122575 Document Number: 99122575. PubMed ID: 9925187. Peripheral-type benzodiazepine receptors in diagnostic subtypes of schizophrenic patients. Wodarz N; Rothenhofer C; Fischer R; Stober G; Kiehl B; Jungkunz G; Riederer P; Klein H E. (Department of Psychiatry, Univ. of Regensburg, Germany.. norbert.wodarz@bzk.uni-regensburg.de) . PSYCHIATRY RESEARCH, (1998 Dec 14) 81 (3) 363-9. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB The peripheral-type benzodiazepine receptor (pBZD-R; also called the omega-3 receptor or the mitochondrial benzodiazepine receptor) seems to play a critical role in the production of neurosteroids, which are able to alter the electrical properties of neuronal membranes and thus the firing patterns of neurons. Putative endogenous ligands are the diazepam-binding inhibitor and its processing products, as well as porphyrins, some of them, in the case of porphyria, are well known to give rise to certain aspects of neuropsychiatric disorders, such as schizophrenic-like symptoms. Previous findings of altered benzodiazepine binding sites in post-mortem brain samples and **platelets** from small samples of schizophrenic patients have been inconclusive. Therefore we investigated characteristic binding parameters (Bmax, Kd) of the granulocytic pBZD-R by using the selective ligand PK11.195 in 53 subjects, fulfilling ICD-10 and DSM-IV criteria of **schizophrenia**. The binding parameters in our total group of 53 schizophrenic patients did not differ from those in healthy subjects. However, Bmax values were significantly reduced in schizophrenic patients with predominantly negative symptoms (residual type) compared to schizophrenic patients with predominantly positive symptoms, i.e. paranoid (-50%) and catatonic subtype (-38%). Moreover, only residual type schizophrenics exhibited a significantly reduced binding capacity compared to healthy subjects (-38%). More studies are warranted to clarify the functional significance of this binding site in the pathogenesis of negative symptoms.

L24 ANSWER 41 OF 160 MEDLINE

1998259958 Document Number: 98259958. PubMed ID: 9597670. **Platelet** 5-HT and plasma cortisol concentrations after dexamethasone suppression test in patients with different time course of

schizophrenia. Jakovljevic M; Muck-Seler D; Pivac N; Crncevic Z. (University Psychiatric Clinic, Clinical Hospital Center, Croatian Institute for Brain Research, Zagreb, Croatia.) NEUROPSYCHOBIOLOGY, (1998) 37 (3) 142-5. Journal code: 7512895. ISSN: 0302-282X. Pub. country: Switzerland. Language: English.

AB **Platelet** 5-HT and plasma cortisol concentrations were determined in 59 schizophrenic patients with different time course of illness before and after dexamethasone suppression test (DST). An abnormal DST (nonsuppression) was observed in 51% of patients. In these patients basal cortisol and **platelet** 5-HT concentrations were higher than in patients with normal DST. After DST, plasma cortisol levels were higher in nonsuppressors with intermittent and intermittent-chronic time course, whereas **platelet** 5-HT concentrations were increased in nonsuppressors with intermittent-chronic time course. The results suggest that schizophrenic patients have dysregulated hypothalamic-pituitary-adrenal axis as shown by a high rate of DST nonsuppression, and that nonsuppressors showed hypercortisolemia and hyperserotonemia independent of the time course of **schizophrenia**. No significant association between DST and time course of the illness was found.

L24 ANSWER 42 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

1998127127 EMBASE MAO inhibitory side effects of neuroleptics and **platelet** serotonin content in schizophrenic patients. Meszaros Z.; Borcsiczky D.; Mate M.; Tarcali J.; Tekes K.; Magyar K.. Z. Meszaros, Department of Pharmacodynamics, Semmelweis University of Medicine, Nagyvarad ter 4, H-1445 Budapest, Hungary. Journal of Neural Transmission, Supplement -/52 (79-85) 1998.
Refs: 21.
ISSN: 0303-6995. CODEN: JNTSD4. Pub. Country: Austria. Language: English. Summary Language: English.

AB In order to study the putative monoamine oxidase (MAO) inhibitory side effect of neuroleptics and simultaneous changes in **platelet** serotonin content both MAO-B activity and serotonin (5-HT) content in **platelets** of 30 healthy volunteers and 50 schizophrenic patients treated with neuroleptics were investigated. Our results have shown significantly lower MAO-B activity (15.26 .+-. 6.81 S.D. vs. 8.63 .+-. 3.82mmol/hour/109 **platelets**) and higher **platelet** 5-HT content (906.19 .+-. 285.33 vs. 1,727.85 .+-. 947.40ng/109 **platelets**) in the schizophrenic group. **Platelet** MAO-B activity was considerably lower in paranoid and residual schizophrenics compared with other patients, however, no difference was found in **platelet** 5-HT content between different subtypes of **schizophrenia**. Various neuroleptic treatments did not produce different effects either on **platelet** serotonin content or **platelet** MAO-B activity.

L24 ANSWER 43 OF 160 MEDLINE

97276030 Document Number: 97276030. PubMed ID: 9129783. Seasonal influence on **platelet** 5-HT levels in patients with recurrent major depression and **schizophrenia**. Jakovljevic M; Muck-Seler D; Pivac N; Ljubicic D; Bujas M; Dodig G. (University Psychiatric Clinic, Clinical Hospital Centre Zagreb, Croatia.) BIOLOGICAL PSYCHIATRY, (1997 May 15) 41 (10) 1028-34. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

AB The influence of seasons on **platelet** serotonin (5-HT) concentration was determined in 88 unipolar depressed and 117 schizophrenic male inpatients, and 90 normal male controls. **Platelet** 5-HT concentrations showed moderate, but insignificant intragroup seasonal variations in healthy controls and in the groups of depressed (psychotic and nonpsychotic) and schizophrenic (positive and negative) patients. In spring, **platelet** 5-HT concentrations were higher in schizophrenic patients than in normal controls or in

depressed patients, while in other seasons **platelet** 5-HT concentrations were not significantly different between the groups. Higher **platelet** 5-HT concentrations were detected in psychotic when compared to nonpsychotic depressed patients in summer, fall, and winter. Increased **platelet** 5-HT concentrations observed in schizophrenic patients with positive symptoms clearly separated these patients from patients with negative **schizophrenia**, especially in spring, summer, and fall. Our results indicate the necessity to match patients with regard to the season of the sampling, and to divide depressed and schizophrenic patients into subtypes.

L24 ANSWER 44 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
97315471 EMBASE Document No.: 1997315471. Amino acids, norharman and serotonergic parameters in **schizophrenia**: Clinical and biochemical effects of treatment with risperidone. Verhoeven W.M.A.; Rijn-Van Den Meijdenberg J.C.C.; Hofma E.; Tuinier S.; Fekkes D.; Pepplinkhuizen L.. W.M.A. Verhoeven, Vincent van Gogh Inst. for Psychiat., Venray, Netherlands. New Trends in Experimental and Clinical Psychiatry 13/2 (117-126) 1997.
Refs: 37.
ISSN: 0393-5310. CODEN: NTEPE7. Pub. Country: Italy. Language: English. Summary Language: English.
AB Interest in the role of 5-hydroxytriptamine (5-HT) and amino acids in **schizophrenia** is based on the one hand upon the observations that in a subgroup of acute psychotic patients disturbances are present in the serine- glycine metabolism and on the other hand that atypical antipsychotics act, among others, via 5-HT mechanisms. In the present study the plasma concentrations of amino acids, norharman and 5-HT parameters were investigated in a sample of 67 schizophrenic patients with either an acute relapsing, stable chronic or residual chronic type of disease. In addition, the acute relapsing group was treated with risperidone and the effects on the biochemical parameters were studied. Comparing the three groups of schizophrenic patients, no differences were found in any of the biochemical parameters. Treatment with risperidone revealed a significant increase of 5- HT concentration in **platelets** and whole blood, only in the group of non- responders. The antipsychotic effects and profile of side effects of risperidone were not different from the observations by others.

L24 ANSWER 45 OF 160 MEDLINE
97355244 Document Number: 97355244. PubMed ID: 9211439. **Platelet** 5-HT levels and hypothalamic-pituitary-adrenal axis activity in schizophrenic patients with positive and negative symptoms. Pivac N; Muck-Seler D; Jakovljevic M. (Laboratory for Molecular Neuropharmacology, Ruder Boskovic Institute, Zagreb, Croatia.. Npivac@olimp.irb.hr) . NEUROPSYCHOBIOLOGY, (1997) 36 (1) 19-21. Journal code: 7512895. ISSN: 0302-282X. Pub. country: Switzerland. Language: English.
AB Hypothalamic-pituitary-adrenal (HPA) axis activity and **platelet** 5-HT concentrations were determined before dexamethasone suppression test (DST) in 80 male schizophrenic patients with predominantly positive or negative symptoms. Significant differences in **platelet** 5-HT and no differences in baseline plasma cortisol concentrations among schizophrenic suppressors and nonsuppressors were found. A similar rate of nonsuppression (56% positive and 53% negative schizophrenic patients) was detected. **Platelet** 5-HT, but not plasma cortisol concentrations, could be used to differentiate positive and negative symptoms of **schizophrenia**.

L24 ANSWER 46 OF 160 SCISEARCH COPYRIGHT 2003 ISI (R)
96:690373 The Genuine Article (R) Number: VE855. MYTHS IN MIGRAINE RESEARCH .1. MIGRAINE AND CLUSTER HEADACHE PATIENTS ARE CHARACTERIZED BY SIGNIFICANT ALTERATIONS IN **PLATELET** MONOAMINE-OXIDASE ACTIVITY.

MOSNAIM A D (Reprint); WOLF M E; CURR M; MOSNAIM J N; FREITAG F G; DIAMOND S. FINCH UNIV HLTH SCI CHICAGO MED SCH, DEPT MOL BIOL & PHARMACOL, N CHICAGO, IL, 00000; LOYOLA UNIV, STRITCH SCH MED, MAYWOOD, IL, 60153; VET AFFAIRS MED CTR, TARD DYSKINESIA PROGRAM, N CHICAGO, IL, 00000; BRANDEIS UNIV, WALTHAM, MA, 02254; DIAMOND HEADACHE CLIN LTD, CHICAGO, IL, 00000; CHICAGO COLL OSTEOPATH MED, DEPT FAMILY MED, DOWNERS GROVE, IL, 00000; COLUMBUS HOSP, DIAMOND CLIN, CHICAGO, IL, 00000; COLUMBUS HOSP, INPATIENT HEADACHE PROGRAM, CHICAGO, IL, 00000. HEADACHE QUARTERLY-CURRENT TREATMENT AND RESEARCH (1996) Vol. 7, No. 3, pp. 225-234. Pub. country: USA. Language: ENGLISH.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective: Review and evaluate the evidence for the proposed association between significant alterations in **platelet** monoamine oxidase (MAO) activity, vulnerability to, and occurrence of migraine and cluster headaches. Reexamine the validity of using **platelet** MAO activity as a biological marker for the differential **diagnosis** of vascular headaches, as well as for a number of other medical conditions.

Data Sources: Major citation indexes and other references sources dating as far back as 1928, when the enzyme MAO was first described.

Conclusions: Significant alterations in **platelet** MAO activity have been reported to be associated with a number of diseases of widely diverse etiologies. They include a variety of neuropsychiatric conditions as well as several nutritional, metabolic, immunological, and cardiovascular disorders. The activity of this enzyme has also been reported to be significantly influenced by other factors such as marijuana drug abuse, premenstrual syndrome, tobacco smoking, age, gender, and the use of psychotropic drugs, eg neuroleptics and antidepressants. It is now generally accepted that we currently do not have a ''disease marker function'' for MAO activity; This is true irrespective of the source of the enzyme (**platelet**, brain, etc). It is also clear that if we are ever going to find such a role for MAO, it will have to be the result of well-designed, preferably multicenter, studies.

L24 ANSWER 47 OF 160 MEDLINE

96397027 Document Number: 96397027. PubMed ID: 8804132. Association study between **schizophrenia** and monoamine oxidase A and B DNA polymorphisms. Coron B; Campion D; Thibaut F; Dollfus S; Preterre P; Langlois S; Vasse T; Moreau V; Martin C; Charbonnier F; Laurent C; Mallet J; Petit M; Frebourg T. (Groupe de Recherche sur la Schizophrenie, Universite de Rouen, Centre Hospitalier Specialise du Rouvray, Sotteville Les Rouen, France.) PSYCHIATRY RESEARCH, (1996 Jun 1) 62 (3) 221-6. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB Monoamine oxidases (MAO) A and B, which are encoded by two distinct genes located on the human X chromosome, are both involved in the oxidative metabolism of dopamine. Decreased levels of **platelet** MAO-B activity has been reported in patients with **schizophrenia** and genetic variation in MAO activity had been proposed as a significant factor in the etiology of this disease. We carried out an association study using two intragenic polymorphisms within the MAO-A and MAO-B genes in 110 schizophrenic patients and 87 control subjects. For each polymorphic marker, no significant difference in allelic frequencies was observed between patients and controls. Nevertheless, a trend toward an association between allele 1 of the MAO-B gene and paranoid **schizophrenia** was found. Our results do not support the hypothesis that inherited variants of MAO genes might play a major role in a genetic predisposition to **schizophrenia**. Since several previous reports found a low MAO-B **platelet** activity in patients with paranoid **schizophrenia**, the identification of polymorphisms related to enzyme activity would be useful.

L24 ANSWER 48 OF 160 SCISEARCH COPYRIGHT 2003 ISI (R)
96:200509 The Genuine Article (R) Number: TZ010. EARLY-ONSET ALCOHOLICS HAVE LOWER CEREBROSPINAL-FLUID 5-HYDROXYINDOLEACETIC ACID LEVELS THAN LATE-ONSET ALCOHOLICS. FILSAIME M L; ECKARDT M J; GEORGE D T; BROWN G L; MEFFORD I; LINNOILA M (Reprint). NIAAA, DIV INTRAMURAL CLIN & BIOL RES, CLIN STUDIES LAB, 10-3C103, 10 CTR DR MSC-1256, BETHESDA, MD, 20892 (Reprint); NIAAA, DIV INTRAMURAL CLIN & BIOL RES, CLIN STUDIES LAB, BETHESDA, MD, 20892; NIMH, CLIN SCI LAB, BETHESDA, MD, 20892. ARCHIVES OF GENERAL PSYCHIATRY (MAR 1996) Vol. 53, No. 3, pp. 211-216. ISSN: 0003-990X . Pub. country: USA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background: We investigated the interrelationships of age at onset of excessive alcohol consumption, family history of alcoholism, psychiatric comorbidity, and cerebrospinal fluid monoamine metabolite concentrations in abstinent, treatment-seeking alcoholics.

Methods: We studied 131 recently abstinent alcoholics. Supervised abstinence was maintained on a research ward at the National Institutes of Health Clinical Center for a minimum of 3 weeks. All alcoholics received a low-monoamine diet for a minimum of 3 days before lumbar puncture. Lumbar punctures were performed in the morning after an overnight fast. Monoamine metabolites and tryptophan in cerebrospinal fluid were quantified with liquid chromatography by means of electrochemical detection. Psychiatric **diagnoses** were established from blind-rated Schedule for Affective Disorders and **Schizophrenia**-Lifetime version interviews administered by a research social worker. Severity and age at onset of excessive alcohol consumption were documented with a structured lifetime drinking history questionnaire and with selected alcoholism screening questionnaires (CAGE and Michigan Alcoholism Screening Test). Family history of alcoholism was obtained from the probands.

Results: A majority of the treatment-seeking, primarily white male alcoholics had a lifetime history of psychiatric disorders other than alcoholism None fulfilled criteria for antisocial personality disorder. Early-onset alcoholics (onset of excessive consumption before 25 years of age) had a more severe course of alcoholism and lower mean cerebrospinal fluid 5-hydroxyindoleacetic acid concentration than late-onset alcoholics. Patients who reported both parents to be alcoholics had particularly low mean cerebrospinal fluid 5-hydroxyindoleacetic acid, homovanillic acid, and tryptophan concentrations.

Conclusion: Among treatment-seeking alcoholics, early age at onset is generally associated with a more severe course of alcoholism and lower cerebrospinal fluid 5-hydroxyindoleacetic acid concentration.

L24 ANSWER 49 OF 160 MEDLINE
97120615 Document Number: 97120615. PubMed ID: 9121621. Effect of age on **platelet** 5-HT concentrations in healthy controls, depressed and schizophrenic patients. Muck-Seler D; Bujas M; Ljubic-Thibal V; Jakovljevic M. (Laboratory of Molecular Neuropharmacology, Ruder Boskovic Institute, Zagreb, Croatia.) NEUROPSYCHOBIOLOGY, (1996) 34 (4) 201-3. Journal code: 7512895. ISSN: 0302-282X. Pub. country: Switzerland. Language: English.

AB The influence of age on **platelet** 5-HT concentrations was investigated in 85 male unipolar depressed inpatients, 113 male schizophrenic inpatients and 81 normal male controls. The correlation coefficients between **platelet** 5-HT concentrations and age within groups were very low and nonsignificant. Our results suggest that higher **platelet** 5-HT content, observed in schizophrenic patients, could not be ascribed to the influence of age.

L24 ANSWER 50 OF 160 MEDLINE DUPLICATE 11
97014341 Document Number: 97014341. PubMed ID: 8861176. **Platelet** [3H]dopamine uptake is differentially affected by neuroleptic drug treatment in **schizophrenia** and schizophreniform disorder. Dean

B; Sundram S; Hill C; Copolov D L. (The Rebecca L. Cooper Research Laboratories, The Mental Health Research Institute of Victoria, Victoria, Australia.) PROGRESS IN NEURO-PSYCHOPHARMACOLOGY AND BIOLOGICAL PSYCHIATRY, (1996 Jan) 20 (1) 45-55. Journal code: 8211617. ISSN: 0278-5846. Pub. country: ENGLAND: United Kingdom. Language: English.

AB 1. The uptake of [3H] dopamine was measured using **platelet**-rich plasma (PRP) from neuroleptic-free subjects and again, in some cases, after the subject had been treated with neuroleptic drugs. 2. There were no differences in [3H]dopamine uptake by PRP in subjects who were or were not mentally ill. 3. After treatment with neuroleptic drugs the Km for **platelet** [3H] dopamine uptake had increased in 76% of subjects with **schizophrenia** and 87% of subjects with schizophreniform disorder. Similarly, the Vmax for **platelet** [3H]dopamine uptake had increased in 81% of the subjects with **schizophrenia** and 86% of the subjects with schizophreniform disorder. 4. By contrast, the Km for **platelet** [3H]dopamine uptake had decreased in 94% of subjects who had a psychoses associated with an illness other than **schizophrenia** or schizophreniform-disorder whilst the Vmax for **platelet** [3H]dopamine uptake also decreased by 94% in these subjects. 5. In subjects with psychoses, **platelet** [(3)H] dopamine uptake is differentially altered during neuroleptic drug treatment depending on **diagnosis**.

L24 ANSWER 51 OF 160 CAPLUS COPYRIGHT 2003 ACS

1995:934131 Document No. 123:337435 Assay for the **diagnosis** of **schizophrenia**. Shinitzky, Meir; Deckmann, Michael (Yeda Research and Development Co., Ltd., Israel; Rycus, Avigail). PCT Int. Appl. WO 9523970 A1 19950908, 19 pp. DESIGNATED STATES: W: AU, BR, CA, JP, US; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1995-US2426 19950228. PRIORITY: IL 1994-108789 19940301; IL 1994-110142 19940628.

AB An immunol. assay for the **diagnosis** of **schizophrenia** in an individual is described. The assay comprises the following steps: (a) a blood sample, a **platelet**-contg. fraction of a blood sample, or a fraction contg. **platelet**-assocd. antibodies (PAA) shed from the **platelets** is obtained from an individual; (b) the sample is contacted with **platelet** antigens fixed to a solid support, and subsequently with an antibody detection system; and (c) the binding pattern of the PAA to the **platelet** antigens is detd. and compared to the binding pattern of a sample obtained from a normal individual. A difference in patterns indicates that the individual has a high likelihood of having **schizophrenia**. The assay is capable of differentiating **schizophrenia** from dementia, as well as from Idiopathic Thrombocytopenia Purpura (ITP), an autoimmune disease directed against a **platelet** antigen.

L24 ANSWER 52 OF 160 CAPLUS COPYRIGHT 2003 ACS

1995:690196 Document No. 123:81605 Fc-less anti-human Ig antibody for **diagnosis** of mental disorders. Shinitsky, Meir (Yeda Research and Development Co. Ltd., Israel; Rycus, Avigail). PCT Int. Appl. WO 9512685 A1 19950511, 16 pp. DESIGNATED STATES: W: AU, CA, JP, US, VN; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1994-US12228 19941027. PRIORITY: IL 1993-107515 19931105.

AB There is described an assay for the **diagnosis** of a mental disorder in an individual, e.g. **schizophrenia** or dementia. A blood sample, a **platelet**-contg. fraction thereof, or a fraction contg. **platelet**-assocd. antibodies (PAA) shed from the **platelets** is withdrawn from the individual to be diagnosed. The withdrawn sample is contacted with an anti-human Ig antibody lacking the Fc domain (Fc-less anti-hIg antibody) and the degree of binding thereof to the PAA is detd. A degree of binding above that found in normal

individuals indicates that diagnosed individual has a high likelihood of having a mental disorder. In example, Fab fragments of rabbit anti-human IgG bound to horseradish peroxidase were prepd. for PAA detn. in blood of schizophrenic patients.

L24 ANSWER 53 OF 160 MEDLINE DUPLICATE 12
95274748 Document Number: 95274748. PubMed ID: 7755113. **Platelet** serotonin-2A receptors: a potential biological marker for suicidal behavior. Pandey G N; Pandey S C; Dwivedi Y; Sharma R P; Janicak P G; Davis J M. (Department of Psychiatry, University of Illinois at Chicago 60612, USA.) AMERICAN JOURNAL OF PSYCHIATRY, (1995 Jun) 152 (6) 850-5. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.

AB OBJECTIVE: Abnormalities in the serotonergic system have been implicated in suicidal behavior. Higher numbers of serotonin-2 (5-HT2) receptors have been reported in the post-mortem brain of suicide victims. In order to further examine the role of 5-HT2A receptors in suicidal behavior, the authors studied 5-HT2A receptors in **platelets** of suicidal and nonsuicidal patients as well as normal comparison subjects. METHOD: 5-HT2A receptor levels were determined by using [125I]LSD as a radioligand in **platelets** obtained from hospitalized psychiatric patients (N = 131) and nonhospitalized normal comparison subjects (N = 40) during a drug-free baseline period. Patients were diagnosed according to DSM-III-R criteria, and suicidal behavior was identified by using the Hamilton Depression Rating Scale. RESULTS: The mean maximum number of binding sites (Bmax) of **platelet** 5-HT2A receptors for all suicidal patients was significantly higher than for nonsuicidal patients or normal comparison subjects. This significant difference remained when subgroups of suicidal patients with depression, **schizophrenia**, schizoaffective disorder, or bipolar illness were compared to the other two subject groups. The higher number of **platelet** 5-HT2A receptors in suicidal patients was independent of **diagnosis**. While there was no significant difference in Bmax between patients with serious suicidal ideation and those who made suicidal attempts, both groups had significantly higher Bmax than normal comparison subjects. CONCLUSIONS: The observed higher number of **platelet** 5-HT2A receptors in suicidal patients is independent of **diagnosis** and appears to be associated with both the brain and the **platelets** of suicidal patients. These results thus suggest the potential usefulness of **platelet** 5-HT2A receptors as a biological marker for identifying suicide-prone patients.

L24 ANSWER 54 OF 160 SCISEARCH COPYRIGHT 2003 ISI (R)
95:718633 The Genuine Article (R) Number: RZ638. MONOAMINE OXIDASES AND ALCOHOLISM .1. STUDIES IN UNRELATED ALCOHOLICS AND NORMAL CONTROLS. PARSIAN A (Reprint); SUAREZ B K; TABAKOFF B; HOFFMAN P; OVCHINNIKOVA L; FISHER L. WASHINGTON UNIV, SCH MED, DEPT PSYCHIAT, 4940 CHILDRENS PL, ST LOUIS, MO, 63110 (Reprint); WASHINGTON UNIV, SCH MED, DEPT GENET, ST LOUIS, MO, 63110; UNIV COLORADO, SCH MED, DEPT PHARMACOL, DENVER, CO, 80262. AMERICAN JOURNAL OF MEDICAL GENETICS (09 OCT 1995) Vol. 60, No. 5, pp. 409-416. ISSN: 0148-7299. Pub. country: USA. Language: ENGLISH.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Low **platelet** MAO activity has been associated with alcoholism, In order to evaluate the role of MAO genes in susceptibility to alcoholism, we have taken a biochemical and molecular genetic approach. The sample consisted of 133 alcoholic probands who were classified by subtypes of alcoholism and 92 normal controls, For those subjects typed for **platelet** MAO activity, alcoholics (N = 74) were found not to differ from the non-alcoholics controls (N = 34), Neither was there a significant difference between type I and type II alcoholics or between either subtype and normal controls. However, we do find significant differences between male and female alcoholics, but not between male and

female controls, The allele frequency distribution for the MAO-A and MAO-B dinucleotide repeats is different between the alcoholic sample (N = 133) and the normal control sample (N = 92), In a two-way analysis of variance of MAO-B activity as a function of the allelic variation of each marker locus and **diagnosis**, there is no evidence for mean differences in activity levels for the different alleles, Our findings do not rule out a role for the MAO-B gene in controlling the enzyme activity because the dinucleotide repeats are located in introns. (C) 1995 Wiley-Liss, Inc.

L24 ANSWER 55 OF 160 MEDLINE
96204675 Document Number: 96204675. PubMed ID: 8624910. [Phospholipase A2 in **schizophrenia**]. Fosfolipaza A2 u schizofrenniho onemocneni. Ripova D; Farska I; Nemcova V; Papezova H. (Psychiatricke centrum Praha.) CESKA A SLOVENSKA PSYCHIATRIE, (1995 Nov) 91 (5) 259-64. Journal code: 9516290. ISSN: 1212-0383. Pub. country: Czech Republic. Language: Czech.
AB The objective of the present work was to evaluate on the basis of the authors' data and data in the literature the role of phospholipase A2 in the pathogenesis of **schizophrenia**. The authors submit available literature pertaining to the problem as well as their own experimental findings. Based on the submitted facts, it cannot be states that phospholipase A2 is a specific marker of **schizophrenia**.

L24 ANSWER 56 OF 160 MEDLINE DUPLICATE 13
95312595 Document Number: 95312595. PubMed ID: 7792339. **Platelet** monoamine oxidase activity and deficit syndrome **schizophrenia**. Samson J A; Gurrera R J; Nisenson L; Schildkraut J J. (Department of Psychiatry, Harvard Medical School, McLean Hospital, Belmont, MA 02178, USA.) PSYCHIATRY RESEARCH, (1995 Jan 31) 56 (1) 25-31. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.
AB Measures of affective flattening that combine self-reported emotional experience with observed affect may identify deficit syndrome patients better than ratings based on observed affect alone. In this study, we examined 23 clinically stable but chronically ill schizophrenic patients, 15 of whom were found to have a deficit syndrome. After exclusion of patients with self-reported depressed mood from the deficit syndrome group, the remaining patients with a deficit syndrome not accompanied by self-reported depressed mood showed a strikingly homogeneous distribution of **platelet** monoamine oxidase activity. Results suggest that inclusion of self-reported emotional experience in clinical definitions of the deficit syndrome will increase the specificity of **diagnosis**.

L24 ANSWER 57 OF 160 MEDLINE DUPLICATE 14
95167163 Document Number: 95167163. PubMed ID: 7863013. GABA-transaminase in brain and blood **platelets**: basic and clinical aspects. Sherif F M. (Department of Pharmacology, University for Medical Sciences, Tripoli, Libya.) PROGRESS IN NEURO-PSYCHOPHARMACOLOGY AND BIOLOGICAL PSYCHIATRY, (1994 Dec) 18 (8) 1219-33. Ref: 96. Journal code: 8211617. ISSN: 0278-5846. Pub. country: ENGLAND: United Kingdom. Language: English.
AB Several lines of evidence suggest that the major inhibitory neuro-transmitter, gamma-aminobutyric acid (GABA) is involved, both directly and indirectly, in the pathogenesis of certain neurological and psychiatric disorders. The main enzyme responsible for GABA catabolism is gamma-aminobutyrate aminotransferase (GABA-T). Inhibition of this enzyme produces a considerable elevation of brain GABA concentrations, and such elevation has been correlated with many pharmacological effects. There seems to be that, as is discussed below, GABA-T activity in the brain and/or blood **platelets** is related to some neuro-psychiatric disorders such as alcoholism, epilepsy and Alzheimer's disease. GABA-T has been identified in the blood **platelets** with similar characteristics to those of brain GABA-T. In this way, studies on GABA-T activity in neuro-psychiatric disorders could be performed to understand, **diagnosis** and treat GABA-related disorders of the central nervous

system (CNS).

L24 ANSWER 58 OF 160 MEDLINE
94255728 Document Number: 94255728. PubMed ID: 7910978. Are there neurochemical indicators of risk for **schizophrenia**?. Csernansky J G; Newcomer J W. (Dept. of Psychiatry, Washington University School of Medicine, St. Louis, MO 63110.) SCHIZOPHRENIA BULLETIN, (1994) 20 (1) 75-88. Ref: 125. Journal code: 0236760. ISSN: 0586-7614. Pub. country: United States. Language: English.
AB The genetic predisposition for certain forms of **schizophrenia** may involve heritable abnormalities in the functioning of neurochemical systems that project to and modulate limbic brain structures. However, with regard to both dopaminergic and serotonergic systems, there is little evidence that either basal cerebrospinal markers or plasma markers predict increased risk for the development of **schizophrenia**. Either their validity as correlates of brain monoamine function is uncertain or they are highly dependent upon clinical state. Both (1) **platelet** and neuroendocrine markers of serotonergic function and (2) an individual's capacity to decrease plasma homovanillic acid concentrations following neuroleptic blockade appear to be less state dependent, and these are worthy of further study as markers of risk for the development of **schizophrenia**.

L24 ANSWER 59 OF 160 MEDLINE DUPLICATE 15
94027476 Document Number: 94027476. PubMed ID: 8214185. Plasma and **platelet** excitatory amino acids in psychiatric disorders. Altamura C A; Mauri M C; Ferrara A; Moro A R; D'Andrea G; Zamberlan F. (Department of Clinical Psychiatry, University of Milan School of Medicine, Italy.) AMERICAN JOURNAL OF PSYCHIATRY, (1993 Nov) 150 (11) 1731-3. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.
AB Plasma and **platelet** levels of excitatory amino acids were measured in 38 psychiatric out-patients and in 19 comparison subjects; the patients had DSM-III-R **diagnoses** of organic mental disorders (N = 3), mood disorders (N = 15), **schizophrenia** (N = 13), and anxiety disorders (N = 7). The glutamate plasma levels were significantly higher in the patients with mood disorders than in the comparison group.

L24 ANSWER 60 OF 160 CAPLUS COPYRIGHT 2003 ACS
1993:18858 Document No. 118:18858 Method of diagnosing or categorizing disorders from biochemical profiles. Matson, Wayne R. (ESA, Inc., USA). PCT Int. Appl. WO 9213273 A1 19920806, 42 pp. DESIGNATED STATES: W: CA, JP, RU; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1992-US375 19920116. PRIORITY: US 1991-643541 19910118; US 1991-649676 19910201.
AB A method for diagnosing disorders in living organisms is disclosed, in which fluid samples from normal and afflicted (abnormal) individuals are analyzed to generate patterns representative of mol. constituents of said samples. A data base of frequency distribution patterns of constituents of samples from organisms having known categories of disorders and controls is created, and the unknown sample anal. is compared for conformity to the frequency distribution patterns. The invention has particular applicability to diagnosing diseases, e.g. Alzheimer's disease, Parkinson's disease, Huntington's disease, **schizophrenia**, progressive supranuclear palsy, amyotrophic lateral sclerosis, and senile dementia. The invention also may be advantageously employed to diagnose diseases such as tumors, carcinomas, cardiovascular abnormalities, and other disorders, or for selection of the therapy based on categories of known vs. unsuccessful outcomes. Moreover, both treatment protocols and new pharmaceuticals may be evaluated. Cerebrospinal fluid samples from patients with Alzheimer's disease, Parkinson's disease, **schizophrenia**, Huntington's disease, and supranuclear palsy and from neurol. normal controls were analyzed by chromatog. and a 16-sensor

electrochem. cell for 38 known components (e.g. adenine, cysteine, tyramine, uric acid, etc.) and for 18 well-defined unknown peaks. Linear and stepwise regression anal. were used in preliminary evaluation of the data and then cluster anal. procedures were performed. The biochem. response of controls or normal individuals was more chaotic then that of disordered individuals. Frequency distribution graphs of Alzheimer's disease and controls were prepd. as well as a plot showing scoring of Alzheimer's vs. control.

L24 ANSWER 61 OF 160 MEDLINE

93002715 Document Number: 93002715. PubMed ID: 1356426. **Platelet** monoamine oxidase in **schizophrenia**: a meta-analysis. Marcolin M A; Davis J M. (Departamento de Psiquiatria, Faculdade de Medicina, Universidade de Sao Paulo, Brazil.) SCHIZOPHRENIA RESEARCH, (1992 Sep) 7 (3) 249-67. Journal code: 8804207. ISSN: 0920-9964. Pub. country: Netherlands. Language: English.

AB We did a meta-analysis on all publications (English and other languages) concerned with **platelet** monoamine oxidase (MAO) in **schizophrenia**. Essentially, when patients were medicated with a neuroleptic, most studies found that schizophrenics had lower **platelet** MAO levels than controls. Administration of neuroleptic lowers MAO levels. MAO levels in drug-free schizophrenics were similar to controls. Only a minority of studies found drug-free schizophrenics had decreased **platelet** MAO levels.

L24 ANSWER 62 OF 160 MEDLINE

92220907 Document Number: 92220907. PubMed ID: 1561286. Dopamine uptake by **platelets** from subjects with **schizophrenia**: a correlation with the delusional state of the patient. Dean B; Kulkarni J; Copolov D L; Shrikanthan P; Malone V; Hill C. (Neurochemistry Section, Mental Health Research Institute of Victoria, Parkville, Australia.) PSYCHIATRY RESEARCH, (1992 Jan) 41 (1) 17-24. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB The uptake of 3H-dopamine by **platelets** from patients with a number of psychiatric disorders has been compared with that by **platelets** from normal volunteers. Overall, 3H-dopamine uptake by **platelet**-rich plasma (PRP) from 25 schizophrenic subjects did not differ from 3H-dopamine uptake by PRP from 22 nonschizophrenic patients and 61 normal volunteers. In the schizophrenic group, however, there was an increased spread of results with seven values falling outside the range of results observed in the control group. Furthermore, of the patients rated, only for the schizophrenic patients was there an inverse correlation between 3H-dopamine uptake by **platelets** and the rating for delusions on the Scale for the Assessment of Positive Symptoms. Thus, 3H-dopamine uptake by **platelet** seems, in some way, to be linked to be delusional state of the patient. Further study of 3H-dopamine uptake by **platelets** is warranted in a larger and more diverse group of patients to determine the significance of altered dopamine uptake by **platelets** from some schizophrenic subjects and the correlation between **platelet** 3H-dopamine uptake and the delusional state of these subjects.

L24 ANSWER 63 OF 160 MEDLINE

93096868 Document Number: 93096868. PubMed ID: 1461943. Monoamine oxidase and cortisol response in depression and **schizophrenia**. Pandey G N; Sharma R P; Janicak P G; Davis J M. (Department of Psychiatry, University of Illinois College of Medicine, Chicago.) PSYCHIATRY RESEARCH, (1992 Oct) 44 (1) 1-8. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB The relationship between hypothalamic-pituitary-adrenal (HPA) axis function and **platelet** monoamine oxidase (MAO) activity was examined in drug-free depressed (n = 32) and schizophrenic (n = 36)

inpatients. HPA function was measured by determining plasma cortisol levels at 8:30 a.m. and 11 p.m. before, and 8:30 a.m., 4 p.m., and 11 p.m. after administration of 1 mg of dexamethasone (DEX). There was a significant correlation between **platelet** MAO activity and all post-DEX cortisol levels (8:30 a.m., 4 a.m., and 11 p.m.) in depressed patients, and MAO activity and pre-DEX cortisol levels (11 p.m.) in schizophrenic patients. MAO activity was significantly higher in depressed DST nonsuppressors than in suppressors, and there were more DST nonsuppressors in high-MAO groups as compared with low-MAO groups. Our results thus suggest a strong relationship between **platelet** MAO activity and HPA function in depressed patients. These biochemical markers are potentially useful in the identification of biochemically and clinically homogeneous subgroups of depressed patients.

L24 ANSWER 64 OF 160 MEDLINE
92089316 Document Number: 92089316. PubMed ID: 1751626. Increased **platelet** membrane lysophosphatidylcholine in **schizophrenia**
. Pangerl A M; Steudle A; Jaroni H W; Rufer R; Gattaz W F. (Central Institute of Mental Health, Unit Neurobiology of Functional Psychoses, Mannheim, Germany.) BIOLOGICAL PSYCHIATRY, (1991 Oct 15) 30 (8) 837-40. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

L24 ANSWER 65 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
92117011 EMBASE Document No.: 1992117011. Basal cortisol, dexamethasone suppression test and **platelet** 5-HT in recurrent (unipolor) major depression, **schizophrenia** and schizoaffective disorder.
Jakovljevic M.; Muck-Seler D.; Kenfelj H.; Plavsic V.; Biocina S.; Kastratovic D.; Ljubicic D.. Yugoslavia. Psychiatria Danubina 3/4 (389-414) 1991.
ISSN: 0353-5053. CODEN: PSYDEI. Pub. Country: Yugoslavia. Language: German. Summary Language: English; German.

L24 ANSWER 66 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
92283517 EMBASE Document No.: 1992283517. **Platelet** autoantibodies in dementia and **schizophrenia**: Possible implication for mental disorders. Shinitzky M.; Deckmann M.; Kessler A.; Sirota P.; Rabbs A.; Elizur A.. Department of Membrane Research, Weizmann Institute of Science,76100 Rehovot, Israel. Annals of the New York Academy of Sciences 621/- (205-217) 1991.
ISSN: 0077-8923. CODEN: ANYAA. Pub. Country: United States. Language: English. Summary Language: English.
AB **Platelets** isolated from blood of demented and schizophrenic patients were found to bear surface antibodies at a considerably higher titer than those found on **platelets** from normal age-matched groups or patients with affective disorders. The **platelet** count in demented and schizophrenic patients correlated inversely with the level of the **platelet** associated antibodies (PAA) which suggested an autoimmune route of opsonization. In most individual cases of dementia or **schizophrenia** PAA and **platelet** count were found to oscillate with time between high PAA-low **platelet** number and low PAA-high **platelet** number in approximately inverse correlation. PAA isolated from demented patients were found to cross-react with **platelets** from normals and with brain tissue from rats. Furthermore, molecular weights of specific brain antigens were identified by binding to PAA. These observations support the possibility that PAA might be implicated in the etiology of some mental dysfunctions associated with dementia and **schizophrenia**.

L24 ANSWER 67 OF 160 MEDLINE
91308030 Document Number: 91308030. PubMed ID: 1854678. Prostaglandin E1 suppression of **platelet** aggregation response in

schizophrenia. Kaiya H. (Department of Neurology and Psychiatry, Gifu University School of Medicine, Japan.) SCHIZOPHRENIA RESEARCH, (1991 Jul-Aug) 5 (1) 67-80. Journal code: 8804207. ISSN: 0920-9964. Pub. country: Netherlands. Language: English.

AB The inhibitory effects of prostaglandin E1 (PGE1) on the **platelet** aggregation response (PAR) to adenosine diphosphate (ADP) in 103 schizophrenics, 55 patients with other mental disorders, and 71 controls were examined. The three groups did not differ in PAR to ADP. However, schizophrenic patients, especially in the acute state, showed a significant reduction in the inhibitory effects of PGE1 on PAR compared to the other two groups. These results suggest PGE1 hyposensitivity exists in some schizophrenic patients, which may result from PGE1 deficiency. As clinical characteristics of the subgroup showing **platelet** PGE1 subsensitivity, relatively successful heterosexual relations, less anergia, and a more severe activation factor on BPRS were identified. Furthermore, the relationship between **platelet** sensitivity to PGE1 and brain morphology, using magnetic resonance imaging on 39 male schizophrenics was examined. Of 11 parameters obtained from MRI measurements, only callosum: brain ratio showed a significant negative correlation with a **platelet** sensitivity to PGE1. The current study suggested existence of a subgroup of **schizophrenia** having **platelet** hyposensitivity and a definite clinical feature as state markers and small corpus callosum as a trait marker.

L24 ANSWER 68 OF 160 MEDLINE

91226584 Document Number: 91226584. PubMed ID: 2027423. [Atypical course of fever in treatment with clozapine]. Atypischer Verlauf einer Fieberentwicklung unter Behandlung mit Clozapin. Hosten K; Gaebel W. (Psychiatrische Klinik und Poliklinik, Freien Universitat Berlin.) NERVENARZT, (1991 Jan) 62 (1) 58-60. Journal code: 0400773. ISSN: 0028-2804. Pub. country: GERMANY: Germany, Federal Republic of. Language: German.

L24 ANSWER 69 OF 160 MEDLINE

91012269 Document Number: 91012269. PubMed ID: 2213639. **Platelet** monoamine oxidase activity and evoked response as predictors of anxiety and depression derived from the content analysis of speech. Sabalesky D A; Demet E M; Chicz-Demet A; Gottschalk L A; Haier R J. (Department of Psychiatry and Human Behavior, College of Medicine, University of California, Irvine 92717.) JOURNAL OF PSYCHIATRIC RESEARCH, (1990) 24 (2) 165-75. Journal code: 0376331. ISSN: 0022-3956. Pub. country: ENGLAND: United Kingdom. Language: English.

AB **Platelet** MAO activity has been reported by several investigators to differentiate **schizophrenia, schizophrenia** related depressive disorders, alcoholism, unipolar and bipolar depression from normal controls. Evoked potentials likewise have differentiated schizophrenic and affective patients. However, the precise relationship between MAO activity, evoked potentials (EP), and psychiatric illness has not been clarified. A possible association between psychopathology and high MAO activity/EP reducing and low MAO activity/EP augmenting has been reported. Such a bidirectionality further confounds results. This study was undertaken to determine the association of psychopathological dimensions found in a group of subjects whose **platelet** MAO activity and evoked responses were obtained two years earlier. Utilizing the Gottschalk-Gleser verbal behavior scales of Anxiety, Depression, Social Alienation-Personal Disorganization and Cognitive Impairment a significant correlation was revealed between low **platelet** MAO activity and high Total Anxiety scale and Shame Anxiety subscale scores. Additionally, a significant correlation was demonstrated between reducing evoked potentials and elevated Death Anxiety, Somatic Concerns, and Total Death and Mutilation Depression subscales scores, combined and separately. Furthermore, a significant positive correlation was found between

augmenting evoked potentials and Overt Hostility Outward scores. No significant correlations were demonstrated between **platelet** MAO activity or evoked potentials and Social Alienation-Personal Disorganization or Cognitive Impairment scores. These findings lend support to the position that biological markers may predict predispositions to anxiety and depression.

L24 ANSWER 70 OF 160 MEDLINE DUPLICATE 16

90192998 Document Number: 90192998. PubMed ID: 1969170. **Platelet** glutathione peroxidase and monoamine oxidase activity in schizophrenics with CT scan abnormalities: relation to psychosocial variables. Buckman T D; Kling A; Sutphin M S; Steinberg A; Eiduson S. (Department of Neurology, Hahnemann University, Philadelphia, PA 19102-1192.) PSYCHIATRY RESEARCH, (1990 Jan) 31 (1) 1-14. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB We have previously reported that the activity in **platelets** of the important antioxidant enzyme glutathione peroxidase (GPx) is inversely correlated with computed tomographic (CT) measures of brain atrophy in a population of patients with chronic **schizophrenia**, suggesting that low GPx may be a vulnerability factor in those schizophrenic patients with structural brain abnormalities. The significance of this finding has now been explored in a larger clinical population by examining the relation of GPx and CT parameters to psychosocial variables and to the activity of **platelet** monoamine oxidase (MAO), which has also been reported to be altered in certain schizophrenic populations. In the present study, low **platelet** GPx and high brain atrophy were found to be associated with DSM-III **diagnoses** of nonparanoid **schizophrenia**, a high degree of chronicity, and a predominance of negative symptoms. Contrary to some literature reports, atrophy also correlated with age and length of illness among the schizophrenic patients, although the contribution of these factors was less than that of low GPx, which was itself not age dependent. The ventricle-brain ratio (VBR) and atrophy were highly correlated in a control group of affective disorder patients, but not in the schizophrenic group, where large VBRs were found predominantly in the DSM-III undifferentiated subgroup. The low-GPx/high-atrophy schizophrenic patients had normal **platelet** MAO levels, and MAO was significantly lower only in the paranoid subgroup, consistent with reported observations. There was no evidence for a neuroleptic-induced effect on either enzyme.

L24 ANSWER 71 OF 160 MEDLINE

90028472 Document Number: 90028472. PubMed ID: 2553139. Accumulation of diacylgylcerol in **platelet** phosphoinositide turnover in **schizophrenia**: a biological marker of good prognosis?. Kaiya H; Nishida A; Imai A; Nakashima S; Nozawa Y. (Department of Neurology and Psychiatry, Gifu University School of Medicine, Japan.) BIOLOGICAL PSYCHIATRY, (1989 Nov) 26 (7) 669-76. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

AB Phosphoinositide (PI) turnover was examined by measuring phosphatidic acid (PA) and diacylglycerol (DG) production following thrombin stimulation in **platelet** membranes from 20 schizophrenic patients, 10 patients with other mental disorders, and 9 normal controls. In 6 of 13 acute schizophrenic patients, DG was not transformed into phosphatidic acid (PA), but accumulated in the **platelets** instead. The abnormal findings persisted for at least 2 months, but then reversed over a long period. Three years later, the patients with abnormal PI turnover had a significantly better outcome than other acute schizophrenic patients.

L24 ANSWER 72 OF 160 MEDLINE

90012809 Document Number: 90012809. PubMed ID: 2796027. Concentrations of beta-phenylethylamine in plasma and plateletes of schizophrenics. Myojin T; Taga C; Tsuji M. (Department of Neuropsychiatry, Kyoto Prefectural

University of Medicine, Japan.) JAPANESE JOURNAL OF PSYCHIATRY AND NEUROLOGY, (1989 Jun) 43 (2) 171-6. Journal code: 8610886. ISSN: 0912-2036. Pub. country: Japan. Language: English.

AB The plasma and **platelet** PEA levels of 20 normal subjects and 17 schizophrenic patients were investigated using a high-performance liquid chromatography. In the normals the mean plasma and **platelet** levels of PEA were 4.9 +/- 1.9 ng/ml and 1.78 +/- 1.01 ng/mg protein, respectively, while in the schizophrenics, those were 12.1 +/- 7.9 ng/ml and 0.77 +/- 0.5 ng/mg protein, respectively. The plasma PEA levels of the schizophrenics were significantly higher than those of the normals, and the **platelet** PEA levels of the schizophrenics were lower than those of the normals.

L24 ANSWER 73 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

89106954 EMBASE Document No.: 1989106954. Biological vulnerability to depression: Replication of MAO and evoked potentials as risk factors. Haier R.J.; Buchsbaum M.S.; DeMet E.; Wu J.. Department of Psychiatry, University of California, Irvine, CA 92717, United States. Neuropsychobiology 20/2 (62-66) 1989. ISSN: 0302-282X. CODEN: NPBYAL. Pub. Country: Switzerland. Language: English. Summary Language: English.

AB In previous work, we have reported that specific combinations of **platelet** (MAO) activity and evoked potential augmenting/reducing (AR) are associated with risk for affective disorders. This new study screened 271 college freshmen solely on MAO and AR and selected a sample with extreme values on both measures. These students were interviewed with the Diagnostic Interview Schedule and they completed a family history questionnaire and psychosocial scales. Following the previous work, the same MAO and AR combinations were related to the frequency of major depression and a family history of psychiatric disorder.

L24 ANSWER 74 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1990:8892 Document No.: BA89:8892. SEROTONIN UPTAKE BY **PLATELETS** OF SUICIDAL AND AGGRESSIVE ADOLESCENT PSYCHIATRIC INPATIENTS. MODAI I; APTER A; MELTZER M; TYANO S; WALEVSKI A; JERUSHALMY Z. GEHAH PSYCHIATR. HOSP., BELINSON MED. CENT., PETAH TIQVA 49 100.. NEUROPSYCHOBIOLOGY, (1989) 21 (1), 9-13. CODEN: NPBYAL. ISSN: 0302-282X. Language: English.

AB Thirty-four adolescent psychiatric inpatients were studied in order to find out whether there is a correlation between serotonin **platelet** uptake (SPU), suicidality and aggression. The patients were divided into four main diagnostic groups according to clinical data: borderline personality disorder, affective disorder (unipolar) including schizoaffective disorder, **schizophrenia** and 'others'. These patients were also characterized by the quantitative symptoms profile from K-SADS scale (Children's Version of the Schedule of Affective Disorders and **Schizophrenia**) and by their behavior: aggression, suicide attempts and violent suicide attempts. In the schizophrenic group, a correlation was found between low Vmax values of SPU and aggressive behavior ($p < 0.05$). In addition, in the 'other' group a correlation was found between low Vmax values of SPU and conduct disorder ($p < 0.05$). On the other hand, in 'other' patients a correlation was found between low Km values of SPU and violent suicide attempt ($p < 0.05$). It is noteworthy that the lowest (20-35%) Vmax values of SPU were found in the patients of the affective group as compared to values of the three other diagnostic groups. These findings are similar to those concerning unipolar depressive adults. It is assumed that there are less binding sites for serotonin on **platelets** of depressive adolescents than was suggested for depressive adults.

L24 ANSWER 75 OF 160 MEDLINE

88239937 Document Number: 88239937. PubMed ID: 3377641. Dopamine metabolism and disposition in schizophrenic patients. Studies using

debrisoquin. Maas J W; Contreras S A; Seleshi E; Bowden C L. (Department of Psychiatry, University of Texas Health Science Center, San Antonio 78284-7792.) ARCHIVES OF GENERAL PSYCHIATRY, (1988 Jun) 45 (6) 553-9. Journal code: 0372435. ISSN: 0003-990X. Pub. country: United States. Language: English.

AB Debrisoquin sulfate, a monoamine oxidase inhibitor that does not enter the brain, was administered to 23 schizophrenic subjects. Plasma, cerebrospinal fluid (CSF), and urine samples were obtained before and during debrisoquin administration and were assayed for their content of norepinephrine and dopamine metabolites, ie, 3-methoxy-4-hydroxyphenylglycol (MHPG), homovanillic acid (HVA), and dihydroxyphenylacetic acid. The severity of the patient's schizophrenic symptoms was also assessed with several types of rating scales. During debrisoquin administration there were significant reductions in plasma, urine, and CSF MHPG levels. Regression analyses suggested that the reduction in CSF MHPG level was probably due to the reduction in plasma MHPG level, which contributes to the CSF MHPG pool. Debrisoquin administration was not associated with changes in CSF HVA level, although it did produce marked reductions in plasma and urinary HVA and dihydroxyphenylacetic acid levels. Significant correlations between plasma and CSF concentrations of HVA were noted during, but not before, debrisoquin administration. Before debrisoquin administration there were trends toward positive relationships between symptom severity and plasma HVA concentrations, which became stronger and statistically significant during debrisoquin administration. These data suggest that debrisoquin may be used as a research tool to create a condition in which measures of HVA in peripheral body fluids reflect dopamine system function and metabolism within the central nervous system.

L24 ANSWER 76 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

88181899 EMBASE Document No.: 1988181899. Determination of **platelet** monoamine oxidase by new continuous spectrophotometric method. Ivanovic I.D.; Majkic-Singh N.. Department of Biochemistry, Faculty of Pharmacy, YU-11000 Belgrade, Yugoslavia. Journal of Clinical Chemistry and Clinical Biochemistry 26/7 (447-451) 1988.
ISSN: 0340-076X. CODEN: JCCBDT. Pub. Country: Germany. Language: English. Summary Language: English.

AB A simple, continuous spectrophotometric method for the determination of tissue monoamine oxidase based on the oxidation of 2,2'-azino-di-(3-ethylbenzthiazoline-6-sulphonate) (ABTS) using peroxidase has already been described (Ivanovic, I. & Majkic-Singh, N. (1986) Fresenius Z. Anal. Chem. 324, 307). In the present study the method is optimized for **platelet** monoamine oxidase assay and applied to healthy persons and schizophrenic patients. The obtained data were statistically analysed. The continuous ABTS method is sensitive, precise (CV below 6.9%) and linear up to 83 U/g protein. Comparision with the endpoint method of Szutowicz et al. (1984) Anal. Biochem. 138, 86-94) gave a good correlation ($r = 0.983$). The reference values for the activity of human **platelet** monoamine oxidase by the new continuous ABTS method are 25 to 42 U/g protein ($x- = 33.2$ U/g protein, CV = 15.5%, $n = 67$). No differences were found between females and males, or between three age groups ranging from 21 to 52 years. The patients with chronic ($n = 76$) or acute ($n = 17$) **schizophrenia** had significantly lower monoamine oxidase activities compared with normal values ($p < 0.005$), which indicates that **platelet** monoamine oxidase can be a possible marker for schizophrenic diseases.

L24 ANSWER 77 OF 160 MEDLINE

89131777 Document Number: 89131777. PubMed ID: 2851919. Imipramine binding in depressive patients diagnosed according to different criteria. Plenge P; Mellerup E T; Gjerris A. (Psychochemistry Institute, Rigshospitalet, Copenhagen, Denmark.) ACTA PSYCHIATRICA SCANDINAVICA,

(1988 Aug) 78 (2) 156-61. Ref: 44. Journal code: 0370364. ISSN: 0001-690X. Pub. country: Denmark. Language: English.

AB 3H-imipramine binding to **platelet** membranes, Bmax and KD, was measured in depressed patients, who were divided into endogenous and non-endogenous depression according to three different criteria, the ICD-9, the Newcastle I and the Newcastle II rating scales. Two groups served as controls, a group of healthy volunteers and a group of psychiatric patients suffering from **schizophrenia** or senile dementia. No significant differences were found in either Bmax or in KD among the different groups of patients and the control groups.

L24 ANSWER 78 OF 160 MEDLINE

88152167 Document Number: 88152167. PubMed ID: 2831076. **Platelet** research in psychiatry. Wirz-Justice A. (Psychiatric University Clinic, Basel, Switzerland.) EXPERIENTIA, (1988 Feb 15) 44 (2) 145-52. Ref: 132. Journal code: 0376547. ISSN: 0014-4754. Pub. country: Switzerland. Language: English.

AB The **platelet** is one of the most researched biological markers in psychiatry. Characteristics of MAO activity, 5-HT uptake, imipramine and alpha 2-adrenergic receptor binding, for example, are similar in **platelet** and CNS. Methodological factors are not negligible, and range from diagnostic specificity and drug effects to the normal physiological variability of age and hormone-related changes, circadian and seasonal rhythms. As yet, there are no clear state or trait **platelet** markers in affective disorders and **schizophrenia** that can be unequivocally used to detect vulnerability to the illness, predict therapeutic response, define clinical diagnostic entities or follow the course of the illness. However, **platelet** markers are increasingly being used in careful studies to monitor psychopharmacological effects (an in vivo assay of all active metabolites), different ligands can be specific markers for certain aspects of a psychiatric illness (e.g. alpha 2-adrenergic receptors and weight loss), and this homogeneous preparation of human cells is an increasingly important tool in studying mechanisms in pathophysiology. More longitudinal studies are required to establish functional relationships between **platelet** variables and psychopathology.

L24 ANSWER 79 OF 160 MEDLINE DUPLICATE 17

88272081 Document Number: 88272081. PubMed ID: 3391497. Binding of imipramine to **platelet** membranes is reduced in panic attacks. Marazziti D; Rotondo A; Placidi G F; Perugi G; Cassano G B; Pacifici G M. (Department of Psychiatry, Medical School, University of Pisa, Italy.) FUNDAMENTAL AND CLINICAL PHARMACOLOGY, (1988) 2 (2) 69-75. Journal code: 8710411. ISSN: 0767-3981. Pub. country: France. Language: English.

AB The binding of imipramine (IMI) to **platelet** membranes was investigated in 13 patients suffering from panic attacks (PA), in 5 patients affected by schizophrenic disorder (S), and in 11 healthy volunteers (V). From 6 volunteers, from 5 patients with panic attacks, and from all the schizophrenic patients, blood samples were collected in the spring, whereas from the others the samples were collected in the autumn. IMI binding was studied according to a protocol provided by the WHO. Binding parameters, the maximum binding capacity (Bmax), and the dissociation constant (Kd) were measured after construction of the Scatchard plot. The differences between V and PA and between V and S were tested by analysis of variance followed by a t-test. Overall and intragroup relationships between Bmax or Kd and **diagnosis** and season were assessed by a 2-way analysis of variance (ANOVA). Bmax (mean +/- SD) was 947 +/- 269 (V), 742 +/- 160 (PA), and 712 +/- 254 (S) fmol/mg protein. V was different from PA (P less than 0.04) and from S (P less than 0.01). Kd (mean +/- SD) was 1.41 +/- 0.6 (V), 1.15 +/- 0.6 (PA), and 0.79 +/- 0.20 (S) nM. V was different from S only (P less than 0.01). Our results show that panic attacks and **schizophrenia** decrease

the binding capacity of IMI in **platelets**. In addition, we found a significant difference between patients and controls only for the samples taken in the spring. No statistically significant difference was detectable between the 2 groups in the autumn samples.(ABSTRACT TRUNCATED AT 250 WORDS)

L24 ANSWER 80 OF 160 MEDLINE DUPLICATE 18
89113505 Document Number: 89113505. PubMed ID: 3217463. EEG sleep, lithium transport, dexamethasone suppression, and monoamine oxidase activity in borderline personality disorder. Lahmeyer H W; Val E; Gaviria F M; Prasad R B; Pandey G N; Rodgers P; Weiler M A; Altman E G. (Dept. of Psychiatry, University of Illinois, Chicago 60680.) PSYCHIATRY RESEARCH, (1988 Jul) 25 (1) 19-30. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.
AB Twenty-one patients who met DSM-III criteria for borderline personality disorder (BPD) and also scored at least 7 on the Diagnostic Interview for Borderlines (DIB) were assessed on four biological markers: electroencephalographic (EEG) sleep, in vitro lithium ratio, **platelet** monoamine oxidase (MAO), and dexamethasone suppression test (DST). REM latency averaged 58.66 (SD 14.39); **platelet** MAO averaged 21.74 (SD 10.33); and lithium ratio was 0.357 (SD 0.139) in the BPD patients. All of those values were significantly abnormal. Many patients had abnormalities on three or four measures. These patients in general had multiple Axis I **diagnoses** from the Diagnostic Interview Schedule (DIS), and these Axis I **diagnoses** tended to produce patient clusters. Patients with a DIS **diagnosis** of **schizophrenia**, mania, hypomania, or schizoaffective mania had elevated lithium, low MAO, and normal EEG sleep, while those patients with coexisting major depression tended to have short rapid eye movement (REM) latency, high REM density, and normal MAO and lithium ratio. Only two patients were nonsuppressors on the DST, confirming recent reports of normal DST results in personality disorders.

L24 ANSWER 81 OF 160 MEDLINE
88074869 Document Number: 88074869. PubMed ID: 2825548. Clinical correlates of **platelet** prostaglandin receptor subsensitivity in **schizophrenia**. Kanof P D; Davidson M; Johns C A; Mohs R C; Davis K L. (Psychiatry Service, Bronx VA Medical Center, NY 10468.) AMERICAN JOURNAL OF PSYCHIATRY, (1987 Dec) 144 (12) 1556-60. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.
AB A diminished cAMP response to prostaglandin E1 (PGE1) in **platelets** from schizophrenic patients has been demonstrated previously. The authors report that among 35 actively psychotic male schizophrenic patients, the **platelet** cAMP response to PGE1 was negatively correlated with global symptom severity and with several indexes of positive symptom severity but not with negative symptom severity. If this subsensitivity of **platelet** PGE receptors extends to brain PGE receptors, schizophrenic patients may have an impairment in the ability of endogenous PGEs to inhibit dopaminergic transmission. Such impairment could have a permissive effect on the production of psychotic symptoms during exacerbations in schizophrenic patients.

L24 ANSWER 82 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
87194686 EMBASE Document No.: 1987194686. A case of mitochondrial encephalomyopathy - mitochondrial myopathy, encephalopathy, lactic acidosis and strokelike episodes (MELAS). Ihara Y.; Namba R.; Demiya M.; et al.. Department of Neurology, Kawasaki Medical School, Kawasaki, Japan. Clinical Neurology 27/8 (969-975) 1987.
CODEN: RISHDJ. Pub. Country: Japan. Language: Japanese. Summary Language: English.
AB A 17-year-old male with mitochondrial encephalomyopathy (MELAS) is presented. He showed convulsions, muscular atrophy, muscular weakness,

visual disturbance, dementia and strokelike episodes, since the age of 10 years. Focal lucencies of brain on computerized tomography (CT) scan appeared and disappeared parallel to strokelike episodes. The patient's mother is short of stature and has dementia. His father has **schizophrenia**. An elder sister of his mother has MELAS. On physical examination, the patient showed a short stature (143 cm) and articular contracture of limbs. Neurological examination showed proximal dominant muscular atrophy and weakness, dementia, amblyopia, concentric contraction of visual field and arteriosclerotic changes of the fundus oculi. No ophthalmoplegia, retinitis pigmentosa, hearing loss, interictal myoclonus, sensory disturbance and cerebellar ataxia were observed. Laboratory examinations showed elevated serum lactate dehydrogenase (type 1 dominant) and serum creatine phosphokinase (type MB dominant). Lactate, pyruvate and the lactate to pyruvate ratio were elevated in the serum and spinal fluid. Metabolic acidosis was evident. Lactate, pyruvate and the lactate to pyruvate ratio in the serum showed a striking increase when strokelike episodes arose. CT scan revealed brain atrophy, calcification of the basal ganglia, transient lucencies and multiple lucencies that do not correspond to territories of vascular supply. These lucencies were characterized with a prolonged T1 relaxation time in nuclear magnetic resonance imaging. Single photon emission CT revealed a decrease of the regional cerebral blood flow. Cerebral angiography was normal. Electrocardiogram was normal. Electroencephalogram showed a generalized spike and wave complex. A giant somatosensory evoked potential and C-response (long loop reflex) were evoked by median nerve stimulation. Latency of pattern reversal visual evoked potential and motor nerve conduction velocity were delayed. In a light microscopic examination of biopsied skeletal muscle (quadriceps femoris), scattered atrophy, central nuclei and ragged-red fibers (Gomori trichrome stain) were evident. Electron microscopic examination of biopsied skeletal muscle and smooth muscle (muscularis mucosae of rectum) revealed abnormal accumulation of mitochondria. Some of these accumulated mitochondria were abnormal in size and shape. High density bodies were also observed. Morphological examination of the sural nerve showed a decrease of the myelinated fibers. Mitochondria within the Schwann cells, the axon, the white blood cells and the blood **platelets** were normal by electron microscopic examination. Activities of pyruvate dehydrogenase complex, pyruvate decarboxylase, pyruvate dehydrogenase phosphatase, .alpha.-ketoglutarate dehydrogenase complex, .alpha.-ketoglutarate decarboxylase, cytochrome c oxydase, succinate cytochrome c reductase, complex II and Km of pyruvate decarboxylase in the skeletal muscle were normal. The concentration of coenzyme Q10 in serum was also normal. This case was classified as MELAS. The cause and mechanism of MELAS, especially strokelike episode, is discussed.

L24 ANSWER 83 OF 160 MEDLINE DUPLICATE 19

87147770 Document Number: 87147770. PubMed ID: 3103151. **Platelet** [3H]imipramine binding in autism and **schizophrenia**. Weizman A; Gonen N; Tyano S; Szekely G A; Rehavi M. PSYCHOPHARMACOLOGY, (1987) 91 (1) 101-3. Journal code: 7608025. ISSN: 0033-3158. Pub. country: GERMANY, WEST: Germany, Federal Republic of. Language: English.

AB [3H]Imipramine binding to **platelet** membranes was evaluated in ten autistics, eight schizophrenics and seven normal controls. The schizophrenics and eight out of the ten autistics were maintained on chronic neuroleptic treatment. **Diagnosis** of autism and **schizophrenia** was established according to the DSM-III criteria. No significant difference in the maximal binding capacity of [3H]imipramine (Bmax) and Kd values could be found among the three groups. It seems that the imipramine binding site is intact both in autism and **schizophrenia**.

L24 ANSWER 84 OF 160 MEDLINE DUPLICATE 20

88069012 Document Number: 88069012. PubMed ID: 3685225. Relationship of auditory hallucinations and paranoia to **platelet** MAO activity in schizophrenics: sex and race interactions. Meltzer H Y; Zureick J L. (Laboratory of Biological Psychiatry, Case Western Reserve University School of Medicine, Cleveland, OH 44106.) PSYCHIATRY RESEARCH, (1987 Oct) 22 (2) 99-109. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Ireland. Language: English.

AB **Platelet** monoamine oxidase (MAO) activity was determined in 37 female and 64 male patients with Research Diagnostic Criteria **diagnoses** of paranoid or undifferentiated **schizophrenia,** or schizoaffective disorder, mainly schizophrenic, and for 71 female and 65 male normal controls (NCs). Female NCs had significantly higher adjusted mean **platelet** MAO activity than male NCs and female, paranoid, nonhallucinating schizophrenics. Male NCs had significantly higher adjusted mean **platelet** MAO activity than male, paranoid, hallucinating schizophrenics. Examination of main and interactive effects of diagnostic subtype, presence/absence of auditory hallucinations, gender, and race within the group of schizophrenic patients revealed no statistically significant main effect but, rather, significant interactive effects of auditory hallucinations with gender, with diagnostic group and gender, and with diagnostic group and race in the prediction of **platelet** MAO activity. The interaction of diagnostic subtype with race and gender in the prediction of **platelet** MAO activity was also statistically significant. In general, significantly decreased **platelet** MAO activity was associated with both paranoid subtype and presence of auditory hallucinations in male and in black schizophrenics; and with paranoid subtype alone in white male schizophrenics. These interactive relationships with **platelet** MAO activity in schizophrenics may account for discrepancies in previous reports of the activity of this enzyme in schizophrenics, and are consistent with reduced **platelet** MAO activity in subgroups of schizophrenics.

L24 ANSWER 85 OF 160 MEDLINE

86322348 Document Number: 86322348. PubMed ID: 3019269. Prostaglandin receptor sensitivity in psychiatric disorders. Kanof P D; Johns C; Davidson M; Siever L J; Coccaro E F; Davis K L. ARCHIVES OF GENERAL PSYCHIATRY, (1986 Oct) 43 (10) 987-93. Journal code: 0372435. ISSN: 0003-990X. Pub. country: United States. Language: English.

AB The cyclic adenosine monophosphate (cAMP) responses to prostaglandin E1 (PGE1) in **platelets** and leukocytes from drug-free schizophrenic patients, depressive patients, and normal controls have been compared. Both schizophrenic and depressive patients had a significantly lower **platelet** cAMP response to PGE1 than controls. The **platelet** cAMP response to PGE1 did not discriminate among exacerbated, remitted, and poor-prognosis schizophrenic patients, or between exacerbated and remitted depressive patients. The cAMP response to PGE1 was negatively correlated with global symptom severity in actively ill schizophrenic patients, but was not correlated with symptom severity in exacerbated depressive patients. The leukocyte cAMP response to PGE1 did not differ among normal controls, schizophrenic patients, and depressive patients. These data indicate that a diminished **platelet** cAMP response to PGE1 may be a marker common to both **schizophrenia** and depression but that this effect does not extend to a cAMP-linked PGE1 receptor on another blood cell type.

L24 ANSWER 86 OF 160 MEDLINE DUPLICATE 21

86211316 Document Number: 86211316. PubMed ID: 3705995. 3H-spiperone binding in **platelet** membranes: a possible biological marker for **schizophrenia**. Sethi B B; Kumar P; Agarwal A K; Seth P K; Trivedi J K. ACTA PSYCHIATRICA SCANDINAVICA, (1986 Feb) 73 (2) 186-90. Journal code: 0370364. ISSN: 0001-690X. Pub. country: Denmark. Language: English.

AB High-affinity-specific 3H-spiperone binding to **platelet** membranes was carried out in 30 schizophrenic patients, without prior antipsychotic medication, fulfilling the Research Diagnostic Criteria, and in 30 matched control subjects. The psychosis was rated on the Modified Brief Psychiatric Rating Scale. Compared with healthy subjects, schizophrenic patients had significantly higher 3H-spiperone binding due to a lower dissociation constant (38%), i.e. increased affinity. No significant difference was observed in the maximum number of binding sites (Bmax) between the two groups. It is our contention that 3H-spiperone binding to **platelets** may be a biological marker for **schizophrenia**.

L24 ANSWER 87 OF 160 MEDLINE DUPLICATE 22
86211313 Document Number: 86211313. PubMed ID: 3705993. A comprehensive study of chronic schizophrenic patients. II: Biological, neuropsychological, and clinical correlates of CT abnormality. Pandurangi A K; Dewan M J; Boucher M; Levy B; Ramachandran T; Bartell K; Bick P A; Phelps B H; Major L. ACTA PSYCHIATRICA SCANDINAVICA, (1986 Feb) 73 (2) 161-71. Journal code: 0370364. ISSN: 0001-690X. Pub. country: Denmark. Language: English.

AB There are numerous reports of lateral cerebral ventricle enlargement on computed tomography (CT) in schizophrenics, but the significance and its relationship to traditional notions of organicity remain unclear. Therefore we studied a subgroup of chronic schizophrenics who had lateral ventriculomegaly (and also cortical hyperdensity) on a battery of relevant biological, neuro-psychological, and clinical parameters such as electroencephalogram (EEG), **platelet** monoamine oxidase (MAO) and serum dopamine-beta-hydroxylase (DBH) activity, the Halstead Reitan Neuropsychological Battery (HRB), premorbid personality adjustment, drug response, positive and negative symptoms, employment history, and family history. Our findings support the notion that there is an "organic" subgroup of **schizophrenia** that has 1) CT structural abnormalities such as lateral ventricle enlargement and cortical hyperdensity; and cerebral dysfunction or deficits as evidenced by 2) an increased incidence of abnormal EEGs and also 3) greater impairment on neuropsychological tests. The biochemical measures, **platelet** MAO and serum DBH activity, nor any of the clinical measures could differentiate between the subgroups. The implications of these findings for the subtyping of **schizophrenia** are discussed.

L24 ANSWER 88 OF 160 MEDLINE
86110021 Document Number: 86110021. PubMed ID: 2867967. Racial and ethnic factors in psychiatric research. Lawson W B. HOSPITAL AND COMMUNITY PSYCHIATRY, (1986 Jan) 37 (1) 50-4. Ref: 73. Journal code: 0040250. ISSN: 0022-1597. Pub. country: United States. Language: English.

AB Although historically research findings about racial and ethnic issues were all too often used to support prevailing concepts of racial inferiority, in recent years racial and ethnic factors have frequently been ignored. However, current findings suggest that racial and ethnic differences exist in the symptom presentations of psychiatric disorders. Significant racial differences have been noted among proposed biological markers for various psychiatric disorders, such as serum creatinine phosphokinase, **platelet** serotonin, and HLA-A2. Racial and ethnic differences in response to psychotropic medication, such as higher blood levels found among Asians, affect dosage requirements and potential side effects. All of these developments underline the importance of considering ethnic and racial factors in psychiatric research.

L24 ANSWER 89 OF 160 MEDLINE
85276808 Document Number: 85276808. PubMed ID: 4025609. **Platelet** MAO in functional psychoses with evidence of brain atrophy. Luchins D J; Arora R C; Meltzer H Y. AMERICAN JOURNAL OF PSYCHIATRY, (1985 Aug) 142 (8)

996-7. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.

L24 ANSWER 90 OF 160 MEDLINE DUPLICATE 23
86096533 Document Number: 86096533. PubMed ID: 4081650. Biological markers in schizotypal personality disorder. Siever L J. SCHIZOPHRENIA BULLETIN, (1985) 11 (4) 564-75. Journal code: 0236760. ISSN: 0586-7614. Pub. country: United States. Language: English.
AB The establishment of the new diagnostic category, Schizotypal Personality Disorder (SPD), has stimulated biological studies of patients with this disorder. Such studies offer the potential of better understanding the **diagnosis** and treatment of SPD as well as more clearly defining the boundaries of the schizophrenic disorders. SPD has been studied in the clinical setting, in family studies of **schizophrenia**, and in the biological high-risk paradigm. In most cases, biological variables associated with **schizophrenia** have been evaluated. Decreased activities of plasma amine oxidase and **platelet** monoamine oxidase have been associated with SPD in the families of schizophrenics and in "biological high-risk" studies. Smooth pursuit eye movement (SPEM) impairment has also been associated with SPD in a "biological high-risk" study of college students. Inferior backward masking performance has been demonstrated in SPD patients in the clinical setting. Other studies using psychophysiological measures have been applied to subjects with psychological characteristics similar to DSM-III SPD and found biological abnormalities similar to those reported in **schizophrenia**. These studies are consistent with the possibility that some individuals with SPD may share common psychobiological abnormalities with schizophrenic individuals and may sharpen our understanding of SPD and its relationship to **schizophrenia**.

L24 ANSWER 91 OF 160 MEDLINE
85281945 Document Number: 85281945. PubMed ID: 3896452. **Schizophrenia**: an amotivational syndrome in men. Lewine R. CANADIAN JOURNAL OF PSYCHIATRY. REVUE CANADIENNE DE PSYCHIATRIE, (1985 Aug) 30 (5) 316-8. Ref: 17. Journal code: 7904187. ISSN: 0706-7437. Pub. country: Canada. Language: English.
AB A series of studies of the phenomenology and biochemistry of **schizophrenia** suggests that the fundamental nature of **schizophrenia** differs in men and women. In men, **schizophrenia** appears to be an amotivational syndrome possibly mediated by dopaminergic underactivity; in women, **schizophrenia** is perhaps best conceptualized as an affective disorder variant.

L24 ANSWER 92 OF 160 MEDLINE DUPLICATE 24
86054031 Document Number: 86054031. PubMed ID: 4064335. **Platelet** monoamine oxidase: specific activity and turnover number in schizophrenics and their families. Summers K M; Andrew B; Gillespie C; Watt D C; Craig I W. CLINICA CHIMICA ACTA, (1985 Nov 15) 152 (3) 289-96. Journal code: 1302422. ISSN: 0009-8981. Pub. country: Netherlands. Language: English.
AB Monoamine oxidase specific activities and molecular turnover numbers have been measured in families with at least two schizophrenic members. Neither measure of monoamine oxidase was different in schizophrenics compared with their first degree relatives. Molecular turnover number was remarkably similar in males and females and when the group was considered by age, **diagnosis**, drug status and family membership. Neither specific activity nor turnover number could be used in risk estimation for the development of **schizophrenia** in members of these families.

L24 ANSWER 93 OF 160 MEDLINE
86073474 Document Number: 86073474. PubMed ID: 4072726. Are schizophrenics with abnormal dexamethasone suppression test results a distinct subgroup?. Dewan M J; Pandurangi A K; Levy B F; Boucher M L;

Major L F. ACTA PSYCHIATRICA SCANDINAVICA, (1985 Sep) 72 (3) 274-7.
Journal code: 0370364. ISSN: 0001-690X. Pub. country: Denmark. Language: English.

AB Nonsuppression on the dexamethasone suppression test (DST) in schizophrenics has been reported by three independent groups. To elucidate the significance of this finding a schizophrenic cohort was tested on a wide range of parameters: computed tomography (CT), electroencephalography (EEG), the Halstead-Reitan Neuropsychological Battery (HRB), **platelet** monoamine oxidase activity (MAO), serum dopamine-beta-hydroxylase levels (DBH), premorbid personality adjustment, response to medication and family history of mental illness. Our results indicate that DST nonsuppressing and DST suppressing schizophrenics are no different on any of these measures, lending support to the notion that DST nonsuppression in schizophrenics is a random and changing event.

L24 ANSWER 94 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

85181467 EMBASE Document No.: 1985181467. **Schizophrenia** at the crossroads: A blueprint for the 80s. Zubin J.; Steinhauer S.R.; Day R.; Van Kammen P.. VA Medical Center, Pittsburgh, PA 15206, United States. Comprehensive Psychiatry 26/3 (217-240) 1985.
CODEN: COPYAV. Pub. Country: United States. Language: English.

AB It is clear that the greatest advance in the 70s has been made in descriptive psychopathology and to prevent this success from becoming a failure in the 80s, we must encourage innovative imagination to overcome the drift toward rigid application of standardized procedures. As for the improved outlook in outcome of **schizophrenia**, the evidence from long-term follow-up studies in Europe indicates that a significant proportion of schizophrenics have only one episode from which they recover and that an even larger proportion show an episodic course of several episodes with final remission. Only a small proportion remain chronically ill continuously but they are the ones who accumulate in our facilities and give **schizophrenia** its undeserved reputation for chronicity. The evidence has also shown that there is considerable doubt whether much of the chronicity that overwhelms our daily practitioners and engenders pessimism is indigenous to **schizophrenia**. There is reason to believe that it may be an artifact engendered by iatrogenic, ecogenic, and nosocomial factors. As for vulnerability markers, there are several potential candidates that have been found in both probands and their unaffected first-degree relatives with frequencies greater than chance expectancy. 1. Cross-over index in reaction time. 2. Smooth pursuit eye movement index. 3. Continuous performance task. 4. Span of apprehension. 5. Dichotic listening with distraction. 6. **Platelet** MAO. We still have to determine whether these markers are vulnerability markers, episode markers, or residual markers. While the specificity of each of these markers for **schizophrenia** is still to be established, the possibility exists that patterns across these markers may serve to identify subgroups of **schizophrenia** and thus reduce the apparent heterogenicity of global **schizophrenia**. The availability of such markers, and the proposed behavioral and chemical challenges for eliciting episode markers as well as the possibility of early warning signs of the imminence of an episode, indicate that the future bodes well for supplementing the current clinical **diagnoses** and treatment with objective indicators that may succeed in reducing the heterogeneity of our nosological categories and reduce the excessive use of neuroleptics and other treatment modalities. These, together with the development of more knowledge about the role of the psychosocial factors in the development of episodes or in their triggering, ought to provide more efficient methods for therapeutic and preventive intervention.

L24 ANSWER 95 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

85106562 EMBASE Document No.: 1985106562. 3H-Imipramine binding sites in **platelets** of hospitalized psychiatric patients. Gentsch C.;

Lichtsteiner M.; Gastpar M.; et al.. Biochemical Laboratory, Department of Psychiatry, University of Basle, CH-4025 Basle, Switzerland. Psychiatry Research 14/3 (177-187) 1985.
CODEN: PSRSDR. Pub. Country: Netherlands. Language: English.

AB The density of **platelet** 3H-imipramine binding sites has been proposed as a biological marker in psychiatry. We report the range of **platelet** 3H-imipramine binding in 55 psychiatric patients and 11 control subjects. All blood samples were withdrawn at 2300 h (on the day of hospital admission for patients). With the use of a slight modification of a previously described 3H-imipramine binding method, a mean B(max) of 1,510 fmole/mg protein (range: 390-5,560; median: 1,450) and a mean K(d) of 2.0 nM (range: 0.6-17.0; median: 1.4) were determined for psychiatric patients. For the controls, a mean B(max) of 1,590 fmole/mg protein (range: 870-2,570; median: 1,440) and a mean K(d) of 1.4 nM (range 0.8-2.4; median 1.4) were determined. When patients were subdivided based on ICD-9 psychiatric **diagnoses**, no significant differences between distinct subgroups of psychiatric patients with respect to B(max) or K(d) values for **platelet** 3H-imipramine binding could be established. Similarly, no significant difference between psychiatric patients and controls was obtained.

L24 ANSWER 96 OF 160 MEDLINE

85257084 Document Number: 85257084. PubMed ID: 2862006. [Blood **platelets**: neuronal model in psychiatric disorders]. La plaquette sanguine: un modele neuronal dans les affections psychiatriques. Dreux C; Launay J M. ENCEPHALE, (1985 Mar-Apr) 11 (2) 57-64. Ref: 51. Journal code: 7505643. ISSN: 0013-7006. Pub. country: France. Language: French.

AB There are some evidences to propose blood **platelets** as a model of bioaminergic neurons. Similarities between **platelets** and neurons are particularly important with respect to serotonin metabolism but now it is possible to extend this model to other neurotransmitters such as dopamine, GABA, glutamate... The reason for these similarities may be due to the common embryonic origin of these two very different cell types. Some changes of **platelet** functions are observed in psychiatric syndromes. For example: serotonin uptake, bioamine storage, enzymatic activities are modified in different types of depression and **schizophrenia**, infantile autism, neurologic diseases (migraine, chorea, Down syndrom). Furthermore, psychotropic drugs also alter the **platelet** functions. Recently, the discovery of neuro-endocrine disorders in psychiatric diseases has led to the proposal of **platelets** as a model in neuro-endocrinology. Some arguments can be developed to support this hypothesis. In biological psychiatry, the **platelet** model seems actually useful essentially in the classification of psychiatric diseases, the management of treatments and the study of new psychotropic drugs. However methodologic difficulties still presently limit the development of this model.

L24 ANSWER 97 OF 160 MEDLINE

85104837 Document Number: 85104837. PubMed ID: 3968053. Does the dexamethasone suppression test have clinical utility in psychiatry?. Baldessarini R J; Arana G W. JOURNAL OF CLINICAL PSYCHIATRY, (1985 Feb) 46 (2 Pt 2) 25-9. Journal code: 7801243. ISSN: 0160-6689. Pub. country: United States. Language: English.

AB The dexamethasone suppression test (DST) has had unprecedented evaluation among biologic tests proposed for application to psychiatric patients. Several techniques for test administration have been devised, including the use of 1 mg of dexamethasone at bedtime, two plasma samples for assay of cortisol (ideally in the afternoon and evening of the following day), and a criterion of greater than 5 micrograms/dl to define nonsuppression. The DST has limited power in differentiating major depression from other acute, severe illnesses but may be useful in comparisons of selected

patient populations with affective features or history, and may also have value in predicting and monitoring treatment response. Experience with the DST encourages the search for additional simple biologic tests to help evaluate psychiatric patients.

L24 ANSWER 98 OF 160 MEDLINE
85082035 Document Number: 85082035. PubMed ID: 3917487. Biological markers for **schizophrenia** and the biological high-risk approach. Siever L J; Coursey R D. JOURNAL OF NERVOUS AND MENTAL DISEASE, (1985 Jan) 173 (1) 4-16. Ref: 131. Journal code: 0375402. ISSN: 0022-3018. Pub. country: United States. Language: English.

AB This review examines two putative biological markers for **schizophrenia**: reduced blood **platelet** monamine oxidase (MAO) activity and impaired smooth pursuit eye movements. Studies of these biological markers among patient samples are presented, including their theoretical background, measurement, genetics, validity, and specificity of the markers for **schizophrenia**, and the artifacts that might lessen their utility. These results are then compared with those from the biological high-risk research strategy, which selects nonpatient volunteers solely on the basis of a deviant marker, and then examines the clinical, psychological, and biological correlates of the marker. The results of these studies suggest that low **platelet** MAO activity is not an adequate marker for **schizophrenia** but is associated with characteristics related to hypomanic behavior and sensation seeking. Smooth pursuit eye tracking impairment, in contrast, seems to be directly associated with **schizophrenia**-related traits, such as the negative symptoms found among chronic schizophrenics, or with the characteristics observed in the biological relatives of schizophrenics. Finally, the implications of these findings are discussed.

L24 ANSWER 99 OF 160 MEDLINE DUPLICATE 25
84167096 Document Number: 84167096. PubMed ID: 6323903. Impaired noradrenergic transmission in **schizophrenia**?. van Kammen D P; Antelman S. LIFE SCIENCES, (1984 Apr 9) 34 (15) 1403-13. Ref: 105. Journal code: 0375521. ISSN: 0024-3205. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Recently, increased brain and spinal fluid (CSF) norepinephrine (NE), and a decreased cAMP response to prostaglandin E1 (PgE1) stimulation of **platelet** NE sensitive adenylcyclase were observed in some schizophrenic patients. Low CSF dopamine-beta-hydroxylase (DBH) activity was related to brain atrophy, whereas high plasma DBH was associated with tardive dyskinesia. Increased NE (in brain and CSF) and 3-methoxy-4-hydroxy-phenylglycol (MHPG) levels and decreased plasma DBH activity in the brain were associated with a **diagnosis** of paranoid **schizophrenia**. Impaired NE transmission in **schizophrenia** may relate to disturbances in the autonomic nervous system, deficits in attention and information processing and to an impaired ability to deal with stress. Although pharmacological studies have suggested a major role for dopamine (DA) in schizophrenic psychosis, this review indicates the need for further exploration of the NE system. Future studies should address the relationship with DA, the autonomic nervous system (ANS), cerebral blood flow, brain metabolism, stress response, negative and prodromal symptoms.

L24 ANSWER 100 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
85039441 EMBASE Document No.: 1985039441. Catecholamines and their receptors in blood: Evidence for alterations in **schizophrenia**. Bondy B.; Ackenheil M.; Birzle W.; et al.. Psychiatrische Klinik der Universitat Munchen, Munchen, Germany. Biological Psychiatry 19/10 (1377-1393) 1984. CODEN: BIPCBF. Pub. Country: United States. Language: English.

AB The simultaneous determination of serum catecholamine (CA) and their

receptors in blood cells offers the possibility of evaluating disturbances of dopamine (DA) and noradrenaline (NA) neuronal systems in man. High-affinity binding sites for 3H-yohimbine in **platelets**, 3H-DHA in granulocytes, and 3H-spiperone in lymphocytes from healthy control persons, unmedicated (n=28), and mediated (n=8) schizophrenics, and from an unmedicated psychiatric control group (n=14) were investigated. Furthermore, the actual concentration of the circulating CA was determined with HPLC-ECD. In unmedicated schizophrenics, as compared with controls, specific binding of 3H-spiperone to lymphocytes was markedly elevated in capacity and less in affinity. For .beta.2 receptors a significant decrease was found in capacity with no change in affinity. The changes in .alpha.2 receptors, viz. a slight decrease in capacity, were less distinct. The concentrations of circulating CA ranged from normal values to a more than threefold increase in NA and DA, whereas adrenaline (A) concentrations were nearly unchanged. No overall change in these data was found in the medicated schizophrenic patients. 3H-Spiperone binding was characteristically increased only in schizophrenics, but did not rise above control data in the nonschizophrenic psychiatric control group. Preliminary family studies suggest that this model could be valuable as a vulnerability marker.

L24 ANSWER 101 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1984:318692 Document No.: BA78:55172. TOWARD A VALIDATION OF THE CONCEPT OF BORDERLINE **SCHIZOPHRENIA**. KHOURI P J. UNIV. TENN. CENT. HEALTH SCI., DEP. PSYCHIATRY, 66 N. PAULINE, SUITE 633, MEMPHIS, TENN. 38105.. COMPR PSYCHIATRY, (1984) 25 (3), 367-371. CODEN: COPYAV. ISSN: 0010-440X. Language: English.

AB The concept of borderline **schizophrenia** has survived in DSM-III [Diagnostic and Statistical Manual of Mental Disorders, III] under the term schizotypal personality disorder. First-generation studies of borderline **schizophrenia** have focused on phenomenological criteria for delineation of the syndrome. Four diagnostic systems were published but no external validation is offered, hence, their use is limited. Two published studies give prevalence rates of 12.7 and 24.5% among 1st-degree relatives of schizophrenics. A 2nd generation of studies focusing on biologic dysfunctions already described in schizophrenic populations but targeting high-risk individuals, like the 1st-degree relatives of schizophrenics, will provide external validation for the **diagnosis** of borderline **schizophrenia**. Six areas for study are suggested: biochemical, brain morphology, psychophysiological, electroencephalographic, neuroendocrine and neuromuscular. A clinicobiologic dissection would provide the biologic underpinning for the **diagnosis** of borderline **schizophrenia**. To date, only **platelet** and plasma MAO [monoamine oxidase] levels among 1st-degree relatives have been studied. The heuristic value of this model is discussed.

L24 ANSWER 102 OF 160 MEDLINE
84248477 Document Number: 84248477. PubMed ID: 6588398. Biological markers and psychosis. McGuffin P. PSYCHOLOGICAL MEDICINE, (1984 May) 14 (2) 255-8. Journal code: 1254142. ISSN: 0033-2917. Pub. country: ENGLAND: United Kingdom. Language: English.

L24 ANSWER 103 OF 160 MEDLINE DUPLICATE 26
85033444 Document Number: 85033444. PubMed ID: 6333507. Blood monoamine oxidases and CT scans in subgroups of chronic schizophrenics. Tachiki K H; Kurtz N; Kling A S; Hullett F J. JOURNAL OF PSYCHIATRIC RESEARCH, (1984) 18 (3) 233-43. Journal code: 0376331. ISSN: 0022-3956. Pub. country: ENGLAND: United Kingdom. Language: English.

AB An assay method for monoamine oxidase (MAO) employing whole blood samples is described. With this method, whole blood samples collected from three subgroups of chronic schizophrenic patients were assayed directly for MAOb

activity without the need for prior isolation of **platelets**. Our results support previous findings from assays with **platelets** that chronic paranoid schizophrenics have decreased blood MAOb activity compared to chronic non-paranoid schizophrenics or normal controls. The level of MAOb activity in the blood of the non-paranoid schizophrenics was also linearly correlated with ventricular size (i.e. ventricular brain ratio, VBR) as determined by CT scan analysis. This correlation was not true for the paranoid group. The MAOb data in conjunction with the VBR data were sufficient to divide without overlap our schizophrenic patient population into two clinically defined subgroups.

L24 ANSWER 104 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
84196005 EMBASE Document No.: 1984196005. Low levels of somatostatin in human CSF mark depressive episodes. Agren H.; Lundqvist G.. Department of Psychiatry, University Hospital, S-751 85 Uppsala, Sweden. Psychoneuroendocrinology 9/3 (233-248) 1984.
CODEN: PSYCDE. Pub. Country: United Kingdom. Language: English.
AB Somatostatin-like immunoreactivity was measured in the cerebrospinal fluid (CSF) of 85 inpatients with current or recent episodes of major depressive disorders, diagnosed according to Research Diagnostic Criteria (RDC) as assessed with the Schedule for Affective Disorders and **Schizophrenia** (SADS). Several biopsychiatric tests were run during the same week of investigation. Results indicate low levels of CSG somatostatin to be a state marker for episodes of depression characterized by sad appearance, feelings of tiredness, insomnia, and subjective inability to acknowledge any external precipitants for the depression. CSF somatostatin was negatively related to **platelet** monoamine oxidase (MAO) activity; MAO activity appeared to account better for the degree of melancholic features than did somatostatin. The ratio between 3-methoxy-4-hydroxyphenylglycol (MHPG) and homovanillic acid (HVA) in CSF also correlated negatively with somatostatin. A positive relationship was noted between CSF xanthine and somatostatin. There was a highly significant curvilinear correlation between CSF somatostatin and serum TSH concentrations, but no correlations between CSF somatostatin and serum GH or prolactin, or with plasma cortisol before or after dexamethasone.

L24 ANSWER 105 OF 160 MEDLINE
84222414 Document Number: 84222414. PubMed ID: 6587413. **Platelet** monoamine oxidase and clinical phenomenology of **schizophrenia**. Baron M; Gruen R; Levitt M; Kane J. PSYCHIATRY RESEARCH, (1984 Mar) 11 (3) 205-9. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Netherlands. Language: English.
AB **Platelet** monoamine oxidase activity (MAO) was determined in 39 unmedicated chronic schizophrenic patients and 88 normal control subjects. **Platelet** MAO activity did not distinguish paranoid from nonparanoid patients or patients who met Taylor and Abrams criteria for narrowly defined **schizophrenia** from other schizophrenics. Enzyme activity was not related to either prognostic scores or age at onset of illness. MAO activity was decreased in patients compared to controls, and was lower in males than in females. Our findings indicate that clinical phenomenology, as defined in the present study, is of limited use in identifying biological subtypes of **schizophrenia** with deviant **platelet** MAO activity.

L24 ANSWER 106 OF 160 MEDLINE
84298714 Document Number: 84298714. PubMed ID: 6591224. Negative symptoms and **platelet** monoamine oxidase activity in male schizophrenic patients. Lewine R J; Meltzer H Y. PSYCHIATRY RESEARCH, (1984 Jun) 12 (2) 99-109. Journal code: 7911385. ISSN: 0165-1781. Pub. country: Netherlands. Language: English.
AB A significant positive correlation was found between negative symptoms and **platelet** monoamine oxidase (MAO) activity in unmedicated male, but

not female, schizophrenic patients. This correlation was significant in split halves of the male patients. There was no indication that the correlation was due to either outliers or medication effects. Male schizophrenic patients with high negative symptom scores had significantly higher mean **platelet** MAO activity than either male normal control subjects or male schizophrenic patients with low negative symptom scores. This finding suggests that the extent of negative symptoms in a population of males could affect whether the schizophrenic subjects will be found to have **platelet** MAO activity which differs from that of normal control subjects. The implications of the correlation between **platelet** MAO activity and negative symptoms for the role of brain MAO activity and two of its substrates, dopamine and serotonin, in the etiology of negative symptoms in male schizophrenic patients are discussed.

L24 ANSWER 107 OF 160 CAPLUS COPYRIGHT 2003 ACS
1983:537732 Document No. 99:137732 Altered **platelet** 3H-imipramine binding in schizo-affective and depressive disorders. Suranyi-Cadotte, Barbara E.; Wood, Paul L.; Schwartz, George; Nair, N. P. Vasavan (Res. Cent., Douglas Hosp., Verdun, QC, H44 1R3, Can.). Biological Psychiatry, 18(8), 923-7 (English) 1983. CODEN: BIPCBF. ISSN: 0006-3223.

AB In blood **platelets** of patients with depression without any medication, the no. of [3H]imipramine-binding sites (Bmax) was below normal whereas the affinity const. (Kd) for the sites was normal. In patients with schizo-affective disorders (all receiving Li and neuroleptics), Bmax was normal whereas Kd was above normal; the increase of Kd was not due to the medication. In medicated schizophrenics and medicated patients with depression, both Bmax and Kd were normal. Thus, Bmax of blood **platelets** can differentiate unmedicated patients with depression from normal controls, schizophrenics, and schizo-affective patients.

L24 ANSWER 108 OF 160 MEDLINE DUPLICATE 27
84000692 Document Number: 84000692. PubMed ID: 6615945. **Platelet** serotonin levels in **schizophrenia**: relationship to race and psychopathology. Jackman H; Luchins D; Meltzer H Y. BIOLOGICAL PSYCHIATRY, (1983 Aug) 18 (8) 887-902. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

AB To explore the possible role of serotonin (5-HT) in the etiology of **schizophrenia**, **platelet** 5-HT concentrations were determined in 41 schizophrenic (and schizoaffective, mainly schizophrenic) patients diagnosed by the RDC and 34 normal controls. There was a significant difference between the patient and control groups with the 16 paranoid, 11 undifferentiated, and 8 schizo-affective depressed patients having significantly higher mean **platelet** 5-HT concentrations than the controls. An analysis of variance considering the effect of race, sex, and **diagnosis** demonstrated a significant difference between black patients and black controls but no significant difference between white patients and white controls. Within the patient sample, **platelet** 5-HT concentrations were positively correlated with severity of auditory hallucinations (on the PSE) and negatively correlated with lack of insight (on the PSE) and conceptual disorganization (on the BPRS). In a subsample of 21 patients, there was no relationship between **platelet** 5-HT and CT findings of either enlarged ventricles or cortical atrophy.

L24 ANSWER 109 OF 160 MEDLINE
83211634 Document Number: 83211634. PubMed ID: 6133830. **Schizophrenia**: a review of diagnostic and biological issues. II. Biological issues. Deutsch S I; Davis K L. HOSPITAL AND COMMUNITY PSYCHIATRY, (1983 May) 34 (5) 423-37. Journal code: 0040250. ISSN: 0022-1597. Pub. country: United States. Language: English.

AB Over the years **schizophrenia** has been the object of an extensive amount of research. In Part I of this paper, published in the April H&CP, the authors discussed research into **diagnosis** and prognosis. In particular, they outlined the studies that have been done on the major diagnostic systems, including the first-rank symptoms, the flexible system, the New Haven **Schizophrenia** Index, the Feighner criteria, the Research Diagnostic Criteria, and the Diagnostic and Statistical Manual of Mental Disorders, third edition. In part II they describe the literature on biological issues in **schizophrenia**. Included in their survey are the studies on neuropathological issues and on genetic and biological hypotheses of etiology and pathogenesis. In particular they discuss the possible roles of dopamine, endorphins and neuropeptides, endogenous psychotomimetics, and viruses in **schizophrenia**.

L24 ANSWER 110 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
83251307 EMBASE Document No.: 1983251307. Influence of hemodialysis on monoamine oxidase kinetics in **platelets** from chronic schizophrenic patients. Wahlund L.O.; Smedby Y.; Saaf J.; et al.. Dep. Psychiatry, Karolinska Inst., St. Goran's Hosp., S-112 81 Stockholm, Sweden. Artificial Organs 7/3 (334-339) 1983.
CODEN: ARORD7. Pub. Country: United States. Language: English.
AB **Platelet** monoamine oxidase (MAO) activity in seven chronic schizophrenic patients undergoing hemodialysis treatment was studied. The treatment was performed in a double-blind crossover design. A significant increase in MAO activity was observed after 5 h of active dialysis, whereas inactive dialysis did not significantly alter the enzyme activity. The increase in MAO activity could be explained by the observed significant decrease in apparent K(m) values for the amine (phenethylamine) studied. During a treatment period of 8 weeks, the MAO activity was not significantly altered with either active or sham dialysis. No relation between clinical ratings and **platelet** MAO activity was observed. Plasma from the seven patients was collected before and after 5 h of active or inactive dialysis and incubated with rat brain mitochondrial MAO (RBM-MAO). No difference was noted in the RBM-MAO-activity-regulating properties of the plasma sampled during either sham or active dialysis.

L24 ANSWER 111 OF 160 MEDLINE
84193916 Document Number: 84193916. PubMed ID: 6144098. The nosology of schizoaffective psychosis. Brockington I F; Meltzer H Y. PSYCHIATRIC DEVELOPMENTS, (1983 Winter) 1 (4) 317-38. Ref: 55. Journal code: 8305469. ISSN: 0262-9283. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The independent status of schizoaffective psychosis is reviewed in relation to 6 hypotheses, using pattern of inheritance, treatment response, course and outcome, and **platelet** 5-HT uptake as discriminating variables. The 'coincidence of 2 diseases' hypothesis would predict an annual frequency of some 2 per 10(8), compared with the observed 2 per 10(5). The 'variant of **schizophrenia**' hypothesis has been supported from patient groupings weighted in favour of chronicity, but little else. The 'variant of affective psychosis' hypothesis has been supported in the manic sub-group by evidence of lithium response, and outcome better than **schizophrenia** (but worse than mania). However in the depressed sub-group, despite responsiveness to ECT, a tendency towards chronicity is observed in a substantial proportion. The 'provisional **diagnosis**' hypothesis is supported by heterogeneity of outcome, and the presence of both **schizophrenia** and affective disorder in families of schizoaffective patients, prompting a distinction between 'mainly schizophrenic' and 'mainly affective' groupings. Evidence for the 'third psychosis' hypothesis is mainly genetic, suggesting a small aetiologically distinct subgroup which breeds true. The authors conclude with an

'interacting process' hypothesis where distinct diseases can cause, or interact to cause, the same symptoms via some final common pathway of psychological dysfunction.

L24 ANSWER 112 OF 160 MEDLINE
83153984 Document Number: 83153984. PubMed ID: 6830934. **Platelet** monoamine oxidase and the growth hormone response to apomorphine in **schizophrenia**. Malas K L; van Kammen D P; deFraites E A; Brown G M; Gold P W. BIOLOGICAL PSYCHIATRY, (1983 Feb) 18 (2) 255-9. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.

L24 ANSWER 113 OF 160 MEDLINE
84193910 Document Number: 84193910. PubMed ID: 6201842. Life at risk: markers of suicidality in depression. Agren H. PSYCHIATRIC DEVELOPMENTS, (1983 Spring) 1 (1) 87-103. Journal code: 8305469. ISSN: 0262-9283. Pub. country: ENGLAND: United Kingdom. Language: English.
AB One hundred and ten patients with Research Diagnostic Criteria (RDC) **diagnoses** of major depressive disorders were assessed for present or recent suicidal ideation and behavior and for suicidal acts earlier in life before current depression using the Schedule for Affective Disorders and **Schizophrenia** (SADS). Suicidal scores were correlated uni- and bivariately with levels of CSF monoamine metabolites (HVA, 5HIAA, MHPG), urinary MHPG, the proportion post-/predexamethasone plasma cortisol at 1100 h, and **platelet** MAO activity (all standardized to same sex, age, height and weight). Results indicate that all 3 monoamine metabolites and their interactions are involved in various aspects of suicidality, at least in unipolar patients. MHPG and 5HIAA (both low or both high) were involved in current or recent suicidal ideation, and low HVA was mainly associated with past potential lethality of suicidal acts. Current hypercortisolism was found in patients that earlier in life had tried to commit dangerous suicides. Bipolar patients (depressives with a history of manic or hypomanic episodes) had earlier in life significantly more, and more dangerous, suicidal attempts than the unipolars.

L24 ANSWER 114 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
82182941 EMBASE Document No.: 1982182941. Human **platelet** monoamine oxidase: A useful enzyme in the study of psychiatric disorders?. Fowler C.J.; Tipton K.F.; MacKay A.V.P.; Youdim M.B.H.. Cent. Rech. Delalande, F-92500 Rueil-Malmaison, France. Neuroscience 7/7 (1577-1594) 1982. CODEN: NRSCDN. Pub. Country: United Kingdom. Language: English.
AB The aim of this review was to point out that, although the study of central monoaminergic processes in some psychiatric diseases may be essential to our further understanding and treatment of these illnesses, the usefulness of **platelet** MAO activity as a monitor may be limited. Not only is the link between **platelet** MAO activity and central monoaminergic turnover not yet proven, but there are also a great many non-psychiatric variables that can affect both **platelet** MAO activity and **platelet** function. However, in view of the ease of collection of blood **platelets** (with respect to more complicated and more invasive procedures such as cerebrospinal fluid collection and to the disadvantages inherent in the use of autopsy material), it is to be hoped that studies on the **platelet** enzyme will continue to be undertaken, but that investigators should constantly be aware of the pitfalls and limitations of this approach.

L24 ANSWER 115 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
82245276 EMBASE Document No.: 1982245276. The effect of oral glucose on **platelet** monoamine oxidase. Karson C.N.; Bridge T.P.; Phelps B.H.; et al.. Adult Psychiatry Branch, Intramural Res. Program, Div. Spec. Ment. Health Res., Natl. Inst. Ment. Health, St. Elizabeths Hosp., Washington, DC, United States. Biological Psychiatry 17/9 (1011-1015) 1982.

CODEN: BIPCBF. Pub. Country: United States. Language: English.

AB **Platelet** monoamine oxidase (MAO) activity was measured serially in seven normal and ten schizophrenic subjects after they ingested 100 g of glucose. There was no decrease in MAO activity in the normals.

L24 ANSWER 116 OF 160 MEDLINE

83058749 Document Number: 83058749. PubMed ID: 6754871. Biological studies of DSM-III psychotic disorders. I. **Platelet** measures and apomorphine-induced growth hormone response. Meltzer H Y; Perline R; Lewine R. JOURNAL OF NERVOUS AND MENTAL DISEASE, (1982 Dec) 170 (12) 758-65. Ref: 59. Journal code: 0375402. ISSN: 0022-3018. Pub. country: United States. Language: English.

AB The relationship between DSM-III **schizophrenia**, major affective disorders, and the psychotic disorders not elsewhere classified (PDNEC) can be explored through studies which attempt to determine whether these disorders can be differentiated from one another and normal controls by biological measures. Preliminary results of an ongoing project which utilizes measures of blood **platelet** monoamine oxidase (MAO), serotonin (5-HT) uptake, and 5-HT content, and the apomorphine-induced increase in growth hormone (GH) to accomplish these goals are reported here. DSM-III major affective disorders (bipolar disorder and major depression) can be differentiated from normal controls by the V max of **platelet** 5-HT. **Platelet** 5-HT V max of bipolar disorder, depressed type, is significantly different from that of **schizophrenia** and PDNEC. Elevated **platelet** 5-HT content is present in black schizophrenic patients compared to black normal controls. **Platelet** MAO was increased in a small group of schizophreniform female patients. There was no difference in the apomorphine-induced GH response between any of the diagnostic groups. If confirmed in a larger series of patients, these results tend to identify the PDNEC more closely with **schizophrenia** than the major affective disorders.

L24 ANSWER 117 OF 160 MEDLINE

83058745 Document Number: 83058745. PubMed ID: 6754869. Biological markers for the schizophrenic and atypical psychoses. Smythies J R. JOURNAL OF NERVOUS AND MENTAL DISEASE, (1982 Dec) 170 (12) 732-6. Ref: 46. Journal code: 0375402. ISSN: 0022-3018. Pub. country: United States. Language: English.

AB This is a review of the present state of knowledge in the area of biological markers that may delineate subpopulations of patients with major psychotic illness. Postmortem studies have revealed that **schizophrenia** is associated with an excess of dopamine (DA) receptors in the limbic system. A more clinically useful adaptation of this approach has been a study of DA D2 receptors in lymphocytes. Studies of monoamine oxidase, dopamine-beta-hydroxylase, and dimethyltryptamine have not fulfilled their early promise nor have the peptides provided useful information as to possible biological markers. Recent studies of the one-carbon cycle enzymes, methionine adenosyltransferase and serine hydroxymethyltransferase, suggest that underactivity of these, particularly the former, may be a reliable clinical marker for a subgroup of schizophrenics. The computerized axial tomography (CAT) scan abnormalities of **schizophrenia** establish useful indices of abnormal cerebral anatomy such as cortical atrophy with enlarged ventricles, cortical asymmetries, and atrophy of the cerebellar vermis. Positron emission tomography studies with 18F 2-deoxyglucose (2DG) have shown that many schizophrenics have a higher 2DG uptake in the occipital and temporal rather than the frontal cortex, thus reversing the normal patterns. The dexamethasone suppression test is a valuable biological marker for certain depressions. It may also be useful in identifying subgroups of the schizoaffective disorders, with some schizoaffectives showing an abnormal affective-like response and others not. These and

other discriminating biological markers are discussed in this report.

L24 ANSWER 118 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
83002442 EMBASE Document No.: 1983002442. Altered **platelet** serotonin uptake kinetics in **schizophrenia** and depression.
Kaplan R.D.; Mann J.J.. Dep. Psychiatry, Cornell Univ. Med. Coll., New York, NY 10021, United States. Life Sciences 31/6 (583-588) 1982.
CODEN: LIFSAK. Pub. Country: United Kingdom. Language: English.
AB **Platelet** uptake of serotonin (5-HT) was studied in drug-free groups of normal controls, depressives and schizophrenics. The Michaelis constant was significantly higher in both the schizophrenics and the depressives than in the controls. The V(max) did not differ significantly between the three groups. Uptake velocity at low substrate concentrations was significantly lower in the schizophrenic group and showed a similar trend in the depressives. There were no significant differences in K(m) or V(max) when depressives were subclassified as unipolar and bipolar. There were no significant correlations between 5-HT uptake kinetics and severity of illness. These findings raise the possibility of a structural defect in the 5-HT reuptake system in the major psychoses that reduces the affinity of the carrier for the amine. This alteration, if present in central serotonergic neurons, may play a role in the etiology and pathogenesis of depression and **schizophrenia**.

L24 ANSWER 119 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
82186190 EMBASE Document No.: 1982186190. [Subgroups of borderline states in psychiatry]. SOUS-GROUPES DANS LES DOMAINE DES ETATS-LIMITES. Stone M.H.. Univ. Connecticut Health Cent., Farmington, CT 06032, United States. Canadian Journal of Psychiatry 27/5 (390-396) 1982.
CODEN: CJPSDF. Pub. Country: Canada. Language: English. Summary Language: French.
AB The currently most popular definitions of 'borderline' are those of Kernberg, Gunderson and Spitzer (now incorporated into the DSM-(III). The Kernberg criteria define a level of function (between 'Neurotic' and Psychotic'); the Gunderson criteria, a more narrowly circumscribed clinical syndrome, phenomenologically distinct from **schizophrenia** and from the psychoneuroses. The DSM-III criteria are derived from these and other sources and define a broad domain that includes the other usages of 'borderline'. Even the narrower definitions of borderline describe a collection of conditions heterogeneous with respect to hereditary, consititutional and psychosocial factors. Genetic, biochemical and clinical research suggests the appropriateness of dividing the borderline domain into a variety of sub-types. The largest proportion of borderline cases are effective (with prominent depressive symptoms; occasionally, with cyclothymic or hypomanic symptoms). Of these, some show strong 'endogenous' features, as well as family pedigrees of manic-depressive illness. This category includes many patients with anorexia nervosa or with agoraphobia. In others, the affective symptoms seem more related to severe psychosocial stresses in early life (including physical abuse, parental deprivation, or incest). Small proportions within the borderline domain are occupied by schizotypal cases (many with hereditary linkage to core **schizophrenia**), or by organic cases (including temporal lobe epilepsy, or minimal brain damage, giving rise to the 'episodic dyscontrol' syndrome). Biochemical and nerophysiological markers that may be useful in distinguishing among the borderline subtypes include measure of **platelet** MAO-activity, of dexamethasone suppression, of R.E.M. latency, motion-sickness susceptibility and of average evoked response to photic stimulation. Attention to subtypes is important in considering optimal treatment for borderline patients. Not all respond to analytically-oriented psychotherapy alone. Those with severe affective symptoms often require antidepressant medication or lithium. Affectively ill borderlines usually have a better prognosis than schizotypals. In cases of episodic dyscontrol, anti-epileptic drugs may be useful.

L24 ANSWER 120 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
82221877 EMBASE Document No.: 1982221877. Serotonin uptake by blood **platelets** of schizophrenic patients. Arora R.C.; Meltzer H.Y.. Dept. Psychiat., Pritzker Sch. Med., Univ. Chicago, Chicago, IL 60637, United States. Psychiatry Research 6/3 (327-333) 1982.
CODEN: PSRSDR. Pub. Country: Netherlands. Language: English.
AB Active uptake of serotonin (5-hydroxytryptamine, 5-HT) by blood **platelets** of acute and chronic schizophrenic patients was compared with that of normal controls. Unlike previous reports, no significant difference in the kinetic parameters (Km and Vmax) of 5-HT uptake between schizophrenic patients and normal controls was observed, although a trend toward decreased V(max) in the acute schizophrenics was present. Since decreased V(max) of **platelet** 5-HT uptake has been found in patients with bipolar, unipolar, and schizoaffective depression, further study of the usefulness of **platelet** 5-HT uptake as a biological marker for major depressive illness is indicated. Abnormalities of 5-HT uptake do not appear to contribute significantly to the increased **platelet** 5-HT levels which have been reported in schizophrenic patients.

L24 ANSWER 121 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
83032718 EMBASE Document No.: 1983032718. Enzyme studies in tardive dyskinesia. I. One year biochemical follow up. Wagner R.L.; Jeste D.V.; Phelps B.H.; Wyatt R.J.. Adult Psychiatry Branch, Div. Spec. Ment. Health, Intramural Res. Program, Natl. Inst. Ment. Health, St. Elizabeths Hosp., Washington, DC, United States. Journal of Clinical Psychopharmacology 2/5 (312-314) 1982.
CODEN: JCPYDR. Pub. Country: United States. Language: English.
AB Over a 1-year period we followed 12 female inpatients with and 13 without persistent tardive dyskinesia. Clinical signs of tardive dyskinesia as well as plasma dopamine-.beta.-hydroxylase and **platelet** monoamine oxidase activities remained stable over time in spite of medication changes. Tardive dyskinesia was associated with higher plasma dopamine-.beta.-hydroxylase and lower monoamine oxidase activities, both initially and at follow-up. In two patients, an apparent elevation in dopamine-.beta.-hydroxylase activity preceded the onset of clinical dyskinesia, suggesting that elevated plasma dopamine-.beta.-hydroxylase activity might be a potential risk marker for the development of tardive dyskinesia.

L24 ANSWER 122 OF 160 MEDLINE DUPLICATE 28
82183136 Document Number: 82183136. PubMed ID: 6122474. Ex uno multi: subtyping the schizophrenic syndrome. Jeste D V; Kleinman J E; Potkin S G; Luchins D J; Weinberger D R. BIOLOGICAL PSYCHIATRY, (1982 Feb) 17 (2) 199-222. Journal code: 0213264. ISSN: 0006-3223. Pub. country: United States. Language: English.
AB We studied 93 chronic schizophrenic inpatients, who met the Research Diagnostic Criteria for **schizophrenia**. Data on a number of historical, epidemiologic, phenomenologic, biochemical, neuropathological, and treatment-response variables were analyzed, using univariate and multivariate statistical analyses. Patients were classified into pairs of subgroups, according to each of the following seven dimensions: (1) ventricle/brain ratio (VBR) assessed on computed tomography scans (normal vs. abnormal); (2) premorbid adjustment (good vs. poor); (3) therapeutic response to neuroleptics (good vs. poor); (4) **platelet** monoamine oxidase (MAO) activity (low vs. high); (5) paranoid features (present vs. absent); (6) tardive dyskinesia (present vs. absence); and (7) hemispheric asymmetry on computed tomography scans (normal vs.. abnormal). These seven dimensions were chosen because earlier studies had shown that the variables involved were operationally definable and were of potential relevance to the subgrouping of schizophrenic patients. Our results

suggested that two biological variables, viz., VBR and **platelet** MAO activity, might be useful in identifying two rather distinct subgroups among chronic schizophrenic patients. A subgroup with large VBR was associated with poor premorbid adjustment, neurological impairment, and poor therapeutic response to neuroleptics, while the subgroup with low **platelet** MAO activity was characterized by the presence of paranoid features and tardive dyskinesia. Possible explanations, implications, and limitations of our findings are discussed.

L24 ANSWER 123 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
82050530 EMBASE Document No.: 1982050530. The effect of lithium on **platelet** monoamine oxidase activity in bipolar and schizoaffective disorders. Meltzer H.Y.; Tueting P.; Jackman H.. Dept. Psychiat., Illinois State Psychiat. Inst., Univ. Chicago Pritzker Sch. Med., 950 East 59th Street, Chicago, IL 60637, United States. British Journal of Psychiatry 140/2 (192-198) 1982.
CODEN: BJPYAJ. Pub. Country: United Kingdom. Language: English.
AB **Platelet** monoamine oxidase (MAO) activity was studied in 33 in-patients with bipolar and schizoaffective disorder who were treated with lithium. **Platelet** MAO activity was found to increase following 10-41 days of lithium treatment compared to a prior drug free period, and the increase was positively correlated with the duration of lithium treatment. The increase in **platelet** MAO activity was not correlated with clinical improvement as measured by the Brief Psychiatric Rating Scale (BPRS) and the Global Assessment Scale (GAS). The number of **platelets** per unit of blood was also significantly correlated with the number of days of lithium treatment. However, the increase in the number of **platelets** in lithium-treated patients was not correlated with the increase in MAO activity and thus appears not to account for it. These results indicate that studies relating **platelet** MAO activity to psychiatric **diagnosis** should be interpreted cautiously if patients are receiving lithium carbonate.

L24 ANSWER 124 OF 160 MEDLINE DUPLICATE 29
83191359 Document Number: 83191359. PubMed ID: 7169931. Reliability, decision rules, and the value of repeated tests. Politser P. MEDICAL DECISION MAKING, (1982) 2 (1) 47-69. Journal code: 8109073. ISSN: 0272-989X. Pub. country: United States. Language: English.
AB While unreliable medical tests often must be repeated, the value of such repetitions may be elusive. This paper attempts to clarify some influences upon the diagnostic usefulness of repeated tests. Reliability is a complex factor whose effect varies with patient heterogeneity and with the decision rule used to resolve conflicts in test results. Two informal rules and one formal statistical method are examined in this paper. In all of these cases, differences in reliability which depend on the disease status of the patient appear to be critical. These differences may alter the value of tests and provide important information that can help us to distinguish between persons with and without a disease. Illustrations of this phenomenon are provided from published data on the hemoccult test in silent colon cancer and the **platelet** monoamine oxidase test in chronic **schizophrenia**. These findings may provide heuristics to refine clinical intuition about the value of repeated tests. They also clarify the need to estimate test reliabilities for both disease and nondisease groups.

L24 ANSWER 125 OF 160 MEDLINE DUPLICATE 30
81231545 Document Number: 81231545. PubMed ID: 7247628. Blood tryptophan metabolism in chronic schizophrenics. Freedman D X; Belendiuk K; Belendiuk G W; Crayton J W. ARCHIVES OF GENERAL PSYCHIATRY, (1981 Jun) 38 (6) 655-9. Journal code: 0372435. ISSN: 0003-990X. Pub. country: United States. Language: English.
AB Concomitant measures of blood indole metabolism were conducted in 33

chronic schizophrenics who showed significantly elevated mean **platelet** serotonin (5-HT) values and lower **platelet** monoamine oxidase (MAO) and plasma amine oxidase activities than normals. When subdivided according to Research Diagnostic Criteria **diagnosis**, the 20 chronic undifferentiated (CU) schizophrenics showed these same deviations from normal; the 13 chronic paranoid (CP) schizophrenics also had significantly higher mean 5-HT values but significantly lower plasma concentrations of total and bound tryptophan. In CP schizophrenics, **platelet** MAO activity, but not plasma amine oxidase activity, was significantly lower than in CU schizophrenics and controls. Hyperserotonemia occurred in 11 of the chronic patients (33%); nine were CU schizophrenics. In the latter, total tryptophan concentration was significantly lowered. Hyperserotonemia was not associated with reduced liver tryptophan pyrrolase activity or **platelet** MAO or plasma amine oxidase activities; rather, it may be a consequence of enhanced tissue tryptophan uptake and utilization.

L24 ANSWER 126 OF 160 MEDLINE

82046174 Document Number: 82046174. PubMed ID: 7295487. Human **platelet** function as a model for investigating the clinical efficacy of chlorpromazine. Youdim M B; Hefez A; Oppenheim B. BRITISH JOURNAL OF CLINICAL PHARMACOLOGY, (1981 Oct) 12 (4) 535-42. Journal code: 7503323. ISSN: 0306-5251. Pub. country: ENGLAND: United Kingdom. Language: English.

AB 1 Enhancement of **platelet** aggregation response (PAR) to 5-hydroxytryptamine (5-HT) in some schizophrenic patients receiving chlorpromazine (CPZ) may provide a biological index for the efficacy of this drug. 2 In a double-blind study 33 schizophrenic patients hospitalized following their first psychotic breakdown were followed up clinically with concurrent assessment of their PAR to 5-HT. The patients followed a standardized treatment schedule with (CPZ) as the sole antipsychotic medication. 3 Twelve patients (Group A) developed an enhanced biphasic 5-HT PAR, within 2-3 weeks and improved clinically by the sixth week. In most cases, the appearance of the enhanced biphasic PAR preceded clinical improvement. Twenty-one patients (Group B) did not have enhanced biphasic PAR to 5-HT by the sixteenth week of treatment. However, twelve subjects from this group showed clinical response to CPZ by the end of this period, while the remaining patients did not improve. 4 The enhanced PAR to 5-HT in Group A discriminated best between good and bad outcome cases when Feighner's research diagnostic criteria were used. We could not confirm the previous reports of **platelet** aggregation response to dopamine in pre- or post-chlorpromazine treatment.

L24 ANSWER 127 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1981:271523 Document No.: BA72:56507. A BIOCHEMICAL STUDY OF TARDIVE DYS KINESIA IN YOUNG MALE PATIENTS. JESTE D V; DELISI L E; ZALCMAN S; WISE C D; PHELPS B H; ROSENBLATT J E; POTKIN S G; BRIDGE T P; WYATT R J. ADULT PSYCHIATRY BRANCH, DIV. OF SPECIAL MENTAL HEALTH RES., INTRAMURAL RES. PROGRAM, NATIONAL INST. OF MENTAL HEALTH, SAINT ELIZABETHS HOSP., WASHINGTON, DC 20032.. PSYCHIATRY RES, (1981) 4 (3), 327-332. CODEN: PSRSDR. ISSN: 0165-1781. Language: English.

AB Based on specific criteria, tardive dyskinesia was diagnosed in 6 of 29 young schizophrenic male inpatients. Several biochemical parameters in these 6 dyskinesia patients were compared with those in 6 matched controls. The patients with dyskinesia had significantly lower **platelet** monoamine oxidase activity and significantly higher plasma dopamine-.beta.-hydroxylase activity as compared with the controls, thus confirming the previous findings in a population of elderly female inpatients. The dyskinetic and nondyskinetic groups did not differ from each other in mean whole blood serotonin concentration and mean serum neuroleptic (thioridazine, mesoridazine haloperidol, fluphenazine, chlorpromazine, thiothixene) concentration as measured with a

radioreceptor binding assay.

L24 ANSWER 128 OF 160 MEDLINE DUPLICATE 31
81192463 Document Number: 81192463. PubMed ID: 6453137. Human **platelet** monoamine oxidase activity in health and disease: a review. Sandler M; Reveley M A; Glover V. JOURNAL OF CLINICAL PATHOLOGY, (1981 Mar) 34 (3) 292-302. Ref: 150. Journal code: 0376601. ISSN: 0021-9746. Pub. country: ENGLAND: United Kingdom. Language: English.
AB The most readily available source of monoamine oxidase in man is the **platelet**, although only the B form of the enzyme is represented in this site. **Platelet** activity is higher in women than in men. The enzyme activity is generally stable and is partly under genetic control. There is some evidence that individuals with low activity have a higher psychiatric morbidity than those with high activity. Despite some negative studies, the consensus of publication dealing with **schizophrenia**, migraine, and alcoholism find that mean **platelet** monoamine oxidase activity in the patient group is lower than in the controls. Values are raised in unipolar depression. Technical differences, or patient or control group heterogeneity, might well account for the absence of unanimity in the literature. A considerable degree of overlap between patient and control values, whatever the clinical **diagnosis**, appears to be the standard finding. Apart from these neuropsychiatric disturbances, **platelet** monoamine oxidase activity is raised in megaloblastic anaemia and reduced in iron deficiency anaemia. Although altered enzyme activity values may be linked to abnormal **platelet** populations in some of the haematological disorders discussed, in general the causes of abnormal **platelet** monoamine oxidase activity are unknown.

L24 ANSWER 129 OF 160 MEDLINE
81160492 Document Number: 81160492. PubMed ID: 6111332. Assessment of anti-psychotic drugs. Mackay A V. BRITISH JOURNAL OF CLINICAL PHARMACOLOGY, (1981 Mar) 11 (3) 225-36. Ref: 84. Journal code: 7503323. ISSN: 0306-5251. Pub. country: ENGLAND: United Kingdom. Language: English.

L24 ANSWER 130 OF 160 MEDLINE
81200004 Document Number: 81200004. PubMed ID: 7232634. **Platelet** monoamine oxidase in an aged general population and elderly chronic schizophrenics [[proceedings]. Bridge T P; Jeste D V; Wise C D; Potkin S G; Phelps B H; Wyatt R J. PSYCHOPHARMACOLOGY BULLETIN, (1981 Jan) 17 (1) 103-4. Journal code: 0101123. ISSN: 0048-5764. Pub. country: United States. Language: English.

L24 ANSWER 131 OF 160 MEDLINE DUPLICATE 32
81143989 Document Number: 81143989. PubMed ID: 7009783. The **schizophrenia** syndrome. Examples of biological tools for subclassification. Wyatt R J; Potkin S G; Kleinman J E; Weinberger D R; Luchins D J; Jeste D V. JOURNAL OF NERVOUS AND MENTAL DISEASE, (1981 Feb) 169 (2) 100-12. Ref: 86. Journal code: 0375402. ISSN: 0022-3018. Pub. country: United States. Language: English.
AB Six biological variables-**platelet** monoamine oxidase activity, urine phenylethylamine concentration, brain norepinephrine concentration, abnormalities on computerized tomography, lateralization asymmetries, and the presence or absence of tardive dyskinesia-are used to discriminate possible biological groups of schizophrenic patients. All variables successfully subclassify patients, some into divisions consistent with phenomological, psychosocial, or biochemical descriptions or hypotheses of **schizophrenia**. None of the measures, however, has sufficiently stood the test of time to be of clinical utility.

L24 ANSWER 132 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
81037713 EMBASE Document No.: 1981037713. Blood **platelets** and

psychiatry. Campbell I.C.. United Kingdom. British Journal of Psychiatry 138/1 (78-80) 1981.
CODEN: BJPYAJ. Pub. Country: United Kingdom. Language: English.

L24 ANSWER 133 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1981:256347 Document No.: BA72:41331. HUMAN **PLATELET** MONO AMINE OXIDASE ACTIVITY IN SUBGROUPS OF SCHIZOPHRENIC DISORDER. RAWAT A K; RAAB E; KOKOTT W. ALCOHOL RESEARCH CENTER C.S. 10002, UNIV. TOLEDO, TOLEDO, OHIO 43699.. RES COMMUN PSYCHOL PSYCHIATRY BEHAV, (1981) 6 (1), 9-20.
CODEN: RCPBDC. ISSN: 0362-2428. Language: English.
AB Hospitalized patients in different subgroups of schizophrenic disorder were studied with respect to biochemical parameters, such as **platelet** monoamine oxidase activity (MAO), plasma dopamine-.beta.-hydroxylase activity and plasma monoamine levels. The majority of previous independent reports have suggested that low **platelet** MAO activity may be associated to genetic disposition to **schizophrenia**. Patients in paranoid, catatonic and undifferentiated subgroups had lower **platelet** MAO activities than controls. The lowest MAO activity was observed in the paranoid schizophrenic subgroup. Schizophrenic patients responding to [drug] treatment started to show an increase in MAO activity as early as 2 wk after the treatment, and the values reached almost control levels by the time the patients were discharged. The patients who did not respond to treatment also did not show a change in the **platelet** MAO activity. A significant difference was not found in the activity of plasma dopamine-hydroxylase between the control and schizophrenic groups. The levels of plasma norepinephrine and dopamine were significantly higher in the schizophrenic subgroups compared with the control group, lending support to the biogenic amine hypothesis of **schizophrenia**. **Platelet** MAO activity and plasma monoamine levels can be helpful tools when used in conjunction with clinical evaluation to assist in the differentiation of **schizophrenia** from other psychotic disorders.

L24 ANSWER 134 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1981:19739 Document No.: BR20:19739. PARANOID **SCHIZOPHRENIA** AND **PLATELET** MONO AMINE OXIDASE ACTIVITY. BARON M; PERLMAN R; LEVITT M. N.Y. STATE PSYCHIATRY INST., 722 W. 168TH ST., NEW YORK, N.Y. 10032, USA.. Am. J. Psychiatry, (1980) 137 (11), 1465-1466. CODEN: AJPSAO. ISSN: 0002-953X. Language: English.

L24 ANSWER 135 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
81069328 EMBASE Document No.: 1981069328. **Platelet** .gamma.-aminobutyric acid-aminotransferase and monoamine oxidase in **schizophrenia**. Reveley M.A.; Gurling H.M.D.; Glass I.; et al.. Bernhard Baron Mem. Res. Lab., Queen Charlotte's Hosp., London W6 0XG, United Kingdom. Neuropharmacology 19/12 (1249-1250) 1980.
CODEN: NEPHBW. Pub. Country: United Kingdom. Language: English.

L24 ANSWER 136 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
81101456 EMBASE Document No.: 1981101456. The activity of blood **platelet** aldehyde reductase in schizophrenic psychoses. Landowski J.; Wontrobski Z.. Klin. Chor. Psych., Akad. Med., Gdansk, Poland. Psychiatria Polska 14/5 (465-470) 1980.
CODEN: PSPOB3. Pub. Country: Poland. Language: Polish. Summary Language: English; Russian.
AB The activity of blood **platelet** aldehyde reductase was determined in 53 patients with **diagnoses** of simple (10), paranoid (36) or catatonic (7) **schizophrenia**. The activity of the enzyme was compared in the three groups and in controls, and found to be statistically significant in the group of paranoid **schizophrenia** . The increase in the activity of that enzyme may lead to a decrease in 3,5-dihydroxyphenylglycol aldehyde concentration and 'inhibit' the

activity of the noradrenergic system, and may thus be responsible for the presence of paranoid symptoms in the patients.

L24 ANSWER 137 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1981:74275 Document No.: BR21:9271. **SCHIZOPHRENIA** AND **PLATELET** MONO AMINE OXIDASE RESEARCH STRATEGIES. BUSCHSBAUM M S; COURSEY R D; MURPHY D L. NIMH, NIH, BUILD 10, ROOM 2N-212, 9000 ROCKVILLE PIKE, BETHESDA, MD. 20205, USA.. Schizophr. Bull., (1980) 6 (2), 375-384. CODEN: SCZBB3. Language: English.

L24 ANSWER 138 OF 160 MEDLINE DUPLICATE 33
81084802 Document Number: 81084802. PubMed ID: 6108742. **Platelet** enzyme abnormalities in neuropsychiatric disease. Farmer P M. ANNALS OF CLINICAL AND LABORATORY SCIENCE, (1980 Jul-Aug) 10 (4) 263-8. Journal code: 0410247. ISSN: 0091-7370. Pub. country: United States. Language: English.
AB Blood **platelets** accumulate, store and release a variety of biogenic amines including norepinephrine, serotonin and dopamine (DA) which are known to act as neurotransmitter substances. **Platelet** monoamine oxidase (MAO) shares many biochemical properties with the mitochondrial MAO present in brain tissue. For these reasons it has been suggested that **platelets** might serve as a diagnostic and research model for nerve cells in a variety of neuropsychiatric diseases. In some patients with **schizophrenia** and manic depressive phychoses, **platelet** MAO activity is significantly decreased. Central nervous system inhibition of MAO could lead to excess accumulation of monoamines in the brain; this would be consistent with the DA hypothesis of **schizophrenia**. Disturbances of monoamines and enzyme kinetics in the hereditary ataxias and in Huntington disease have been described, but these findings are unproven and controversal. If **platelet** models for human neuropsychiatric disease can be established, they will be immensely important in preclinical **diagnosis**, therapy and genetic counseling.

L24 ANSWER 139 OF 160 MEDLINE
80191772 Document Number: 80191772. PubMed ID: 7375851. Monoamine oxidase in **schizophrenia**: an overview. Wyatt R J; Potkin S G; Bridge T P; Phelps B H; Wise C D. SCHIZOPHRENIA BULLETIN, (1980) 6 (2) 199-207. Journal code: 0236760. ISSN: 0586-7614. Pub. country: United States. Language: English.
AB A brief history and summary of studies designed to elucidate the role of monoamine oxidase (MAO) in **schizophrenia** are presented. The majority of these studies have reported a decrease in the **platelet** enzyme activity of chronic schizophrenic patients when compared to controls. Difficulties encountered when comparing MAO activity measured in different patient populations are also considered. Finally, the significance of decreased **platelet** MAO activity is discussed with respect to its possible etiological role in some forms of **schizophrenia**.

L24 ANSWER 140 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 34
79254694 EMBASE Document No.: 1979254694. Biology of **schizophrenia** subtypes: A review and proposal for method of study. Meltzer H.Y.. Dept. Psychiat., Univ. Chicago Pritzker Sch. Med., Chicago, Ill. 60637, United States. Schizophrenia Bulletin 5/3 (460-479) 1979.
CODEN: SCZBB3. Pub. Country: United States. Language: English.
AB This article describes two assumptions that are currently operative in biological research in **schizophrenia**. First, it is assumed that etiologic heterogeneity is present in this group of patients, even within patient cohorts diagnosed by precise, narrow research criteria. Secondly, it is assumed that overlap may exist with regard to specific biological abnormalities between schizophrenics and patients who satisfy research

diagnostic criteria for other psychiatric disease entities. These assumptions suggest a need to relate putative biological abnormalities in schizophrenics to an array of historical, phenomenological, treatment response, long-term outcome, and family data in order to identify schizophrenic subtypes. Concern with biochemical abnormalities as 'markers' for **schizophrenia** should be supplemented by efforts to demonstrate that the abnormality is relevant to the psychopathology of the perhaps small subgroup of patients in whom it is found. The following areas of research are reviewed for evidence of any usefulness in identifying schizophrenic subtypes: dopaminergic abnormalities; studies of other neurotransmitters or neuromodulators, including peptides; neuroendocrine studies, **platelet** monoamine oxidase (MAO) activity studies; autoimmune phenomena; and HLA antigens. The importance of genetic studies for biochemical research in **schizophrenia** is emphasized.

L24 ANSWER 141 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1979:228983 Document No.: BA68:31487. **PLATELET** MONO AMINE OXIDASE ACTIVITY IN **SCHIZOPHRENIA** A REVIEW OF THE DATA. WYATT R J; POTKIN S G; MURPHY D L. LAB. CLIN. PSYCHOPHARMACOL., DIV. SPEC. MENT. HEALTH RES., WILLIAM A. WHITE BUILD., ROOM 536, ST. ELIZABETHS HOSP., WASHINGTON, D.C. 20032, USA.. AM J PSYCHIATRY, (1979) 136 (4A), 377-385. CODEN: AJPSAO. ISSN: 0002-953X. Language: English.

AB Studies of **platelet** and white cell monoamine oxidase (MAO) activity in schizophrenic patients were reviewed. The data on acute schizophrenic patients remain inconclusive. Review of 26 reports of chronic schizophrenic patients leaves little doubt that there is a subgroup in which the enzyme activity is decreased. Despite the strong association of decreased MAO activity and chronic **schizophrenia**, the etiological relationship of low **platelet** MAO activity to **schizophrenia** was not demonstrated. More complete diagnostic descriptions will shed light on precisely which patients have lowered MAO activity. Further metabolic investigations with such patients are needed to determine the physiological significance of this phenomenon.

L24 ANSWER 142 OF 160 MEDLINE DUPLICATE 35
79188469 Document Number: 79188469. PubMed ID: 444787. **Platelet** MAO activity and evoked potentials in the identification of subjects biologically at risk for psychiatric disorders. Coursey R D; Buchsbaum M S; Murphy D L. BRITISH JOURNAL OF PSYCHIATRY, (1979 Apr) 134 372-81. Journal code: 0342367. ISSN: 0007-1250. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Individuals potentially at risk for psychiatric disorders were identified by screening 375 college student volunteers for low **platelet** monoamine oxidase (MAO) activity levels. The lower and upper 10% in MAO activity were administered a personal and family history interview, psychological tests and average evoked response (AER) electroencephalographic procedures. Results indicated that low MAO males and females were socially more active, had more psychiatric contact, and had relatives who were psychiatrically more disturbed than high MAO subjects. Low MAO males had more convictions, experimented more with illegal drugs and had elevated scores on the MMPI. AER criteria further defined a high risk group of low MAO-AER augmenters which had more suicides among their relatives and higher scores on the **schizophrenia** scale of the MMPI.

L24 ANSWER 143 OF 160 MEDLINE
78156311 Document Number: 78156311. PubMed ID: 643045. Monoamine oxidase in **schizophrenia**. Anonymous. NEW ENGLAND JOURNAL OF MEDICINE, (1978 May 18) 298 (20) 1150-2. Journal code: 0255562. ISSN: 0028-4793. Pub. country: United States. Language: English.

L24 ANSWER 144 OF 160 MEDLINE
79038309 Document Number: 79038309. PubMed ID: 706910. [Decrease in the thrombocyte monoamine oxidase activity of schizophrenic patients]. Snizhenie aktivnosti monoaminoksidazy trombotsitov bol'nykh shizofreniei. Kurilova I I; Lideman R R. ZHURNAL NEVROPATOLOGII I PSIKHIATRII IMENI S. S. KORSAKOVA, (1978) 78 (9) 1351-4. Journal code: 8710066. ISSN: 0044-4588. Pub. country: USSR. Language: Russian.

AB The authors measured the monoaminoxidase activity in thrombocytes of 28 schizophrenic patients and in 36 normal donors. The monoamine oxidase activity in schizophrenic patients was decreased by 14% and in the subgroup of patients with continuous **schizophrenia** by 19.5% as compared to that of normals. The monoamine oxidase activity in schizophrenic female patients did not significantly differ from the norm, while the male patients differed highly significantly from the normal. The decrease of monoamine oxidase activity correlated with the level of negative disorders (r = 0,71; P less than 0,01). A correlation with positive disorders was not noted.

L24 ANSWER 145 OF 160 MEDLINE DUPLICATE 36
79020405 Document Number: 79020405. PubMed ID: 697538. Activities of types A and B MAO and catechol-o-methyltransferase in blood cells and skin fibroblasts of normal and chronic schizophrenic subjects. Groshong R; Baldessarini R J; Gibson D A; Lipinski J F; Axelrod D; Pope A. ARCHIVES OF GENERAL PSYCHIATRY, (1978 Oct) 35 (10) 1198-1205. Journal code: 0372435. ISSN: 0003-990X. Pub. country: United States. Language: English.

AB We assayed activities of monoamine oxidase (MAO) type B in blood **platelets** and type A (and B) in fibroblasts cultured from punch biopsy specimens of skin, as well as of catechol-O-methyltransferase (COMT) in erythrocytes and fibroblasts. Fibroblasts contained moderate amounts of both forms of MAO (types A and B) found in human brain and large amounts of COMT activity. Activities of both enzymes correlated poorly between fibroblasts and blood cells. Comparing carefully diagnosed chronic schizophrenics with age-matched normal young men, we found no difference in these biochemical variables, nor could we distinguish patients with paranoid symptoms. In contrast, we confirmed markedly lower MAO activities in **platelet** samples from chronic patients provided by colleagues at the National Institute of Mental Health. Results concerning MAO and COMT activities are now sufficiently inconsistently characteristic of schizophrenics as to question their clinical applicability and to indicate a need for further critical evaluation, with special attention to **diagnosis**, matching of subjects, and effects of possible spurious environmental variables.

L24 ANSWER 146 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
79076599 EMBASE Document No.: 1979076599. Differences in **platelet** monoamine oxidase activity in subgroups of schizophrenic and depressive disorders. Orsulak P.J.; Schildkraut J.J.; Schatzberg A.F.; Herzog J.M.. Dept. Psychiat., Harvard Med. Sch., Boston, Mass., United States. Biological Psychiatry 13/6 (637-647) 1978.
CODEN: BIPCBF. Pub. Country: United States. Language: English.

AB **Platelet** monoamine oxidase (MAO) activity was measured in patients with nonaffective schizophrenic disorders (i.e., without prominent symptoms of depressions or manias), and in patients with **schizophrenia**-related depressions. MAO activity was significantly lower than control values in a subgroup of 16 patients with nonaffective schizophrenic disorders (most of whom were paranoid) characterized by the presence of auditory hallucinations and delusions. **Platelet** MAO activity was not reduced in 16 other nonaffective schizophrenic patients without auditory hallucinations. **Platelet** MAO activity was significantly higher than control values in a group of 8 depressed patients with **schizophrenia**-related depressions characterized by the presence of chronic asocial, eccentric, or bizarre behavior. These

findings of differences in **platelet** MAO activity in clinically defined subgroups of nonaffective schizophrenic disorders and the **schizophrenia**-related depressive disorders may help to account for some of the discrepancies in findings among the various studies of **platelet** MAO activity in schizophrenic and affective disorders.

L24 ANSWER 147 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
79159813 EMBASE Document No.: 1979159813. Effects of a selective inhibitor of type A monoamine oxidase - Lilly 51641 - on behavior, sleep and circadian rhythms in depressed and schizophrenic patients. Carman J.S.; Gillin J.C.; Murphy D.L.; et al.. Dept. Psychiat., Univ. Alabama Med. Cent., Univ. Stat., Birmingham, Ala. 35294, United States. Communications in Psychopharmacology 2/6 (513-523) 1978.
CODEN: CPSZDP. Pub. Country: United Kingdom. Language: English.
AB Lilly 51641 [N-(2-chlorophenoxyethyl)-cyclopropylamine] a selective inhibitor of Type A monoamine oxidase (MAO) activity, was given for 9 to 73 days to eight inpatients with different primary psychiatric **diagnoses**. After two weeks treatment at daily doses exceeding 200 mg by mouth, this patient group exhibited greater than 90% mean reduction in plasma amine oxidase activity, which persisted for three weeks following discontinuation of the drug. Consistent reductions in **platelet** MAO were also noted, although the magnitude was less than 25% and such reductions persisted for less than one week following discontinuation of active drug. All 7 patients receiving active drug for longer than two weeks or at daily doses exceeding 200 mg by mouth exhibited significant and substantial mean increases in ratings of agitation or mania. The three female patients seemed especially vulnerable to such excitation during the week prior to their menses. The only two patients with primary affective illness - one patient with unipolar and one with bipolar depression - had the lowest baseline **platelet** MAO activities, and showed dramatic antidepressant responses on the drug. In contrast, two schizo-affective patients showed either decreased agitation or increased depression during treatment with daily doses of 50 to 150 mg orally. The one schizophrenic and one schizo-affective patient with highest baseline **platelet** MAO were the only two patients who exhibited an increase in psychosis on active drug. Gross fragmentation of nocturnal sleep and daytime drowsiness were seen in all patients. The circadian rhythm in body temperature decreased in amplitude on the drug. All patients developed postural hypotension on this drug - a side effect which was managed adequately by dosage reduction and temporary bedrest.

L24 ANSWER 148 OF 160 MEDLINE
78071540 Document Number: 78071540. PubMed ID: 619236. Are paranoid schizophrenics biologically different from other schizophrenics?. Potkin S G; Cannon H E; Murphy D L; Wyatt R J. NEW ENGLAND JOURNAL OF MEDICINE, (1978 Jan 12) 298 (2) 61-6. Journal code: 0255562. ISSN: 0028-4793. Pub. country: United States. Language: English.
AB Two studies were undertaken to verify the presence of lowered **platelet** monoamine oxidase activity in chronic **schizophrenia**. In the first study, a retrospective chart analysis, the mean **platelet** activity of patients with chronic **schizophrenia** (7.73+/-0.64 nmol of benzylaldehyde product per 10(8) **platelets** per hour [S.E.M.]) differed significantly from that of normal controls (12.13+/-0.2, P less than 0.001). Chronic paranoid schizophrenics (4.81+/-0.46) differed significantly from chronic nonparanoid schizophrenics (8.6+/-0.75, P less than 0.03). A separate prospective study confirmed significantly lower values for monoamine oxidase activity in chronic schizophrenic patients diagnosed as paranoid (5.97+/-1.17) or as having secondary paranoid features (6.28+/-0.71) as compared to chronic nonparanoid schizophrenics (9.81+/-0.87, P less than 0.001). Chronic paranoid **schizophrenia** may be a separate disorder from the other chronic forms of **schizophrenia**, and this

difference may be related, at least in part, to biochemical characteristics.

L24 ANSWER 149 OF 160 MEDLINE

77154958 Document Number: 77154958. PubMed ID: 848577. **Platelet** monoamine oxidase activity in schizophrenic patients. Becker R E; Shaskan E G. AMERICAN JOURNAL OF PSYCHIATRY, (1977 May) 134 (5) 512-7. Journal code: 0370512. ISSN: 0002-953X. Pub. country: United States. Language: English.

AB The authors studied **platelet** monoamine oxidase (MAO) activity in 29 schizophrenic inpatients and 26 schizophrenic outpatients during a 4-week double-blind trial of chlorpromazine with imipramine or thiothixene with placebo. The found that significantly more schizophrenic patients than normal control subjects had low **platelet** MAO activity after 4 weeks. Outpatients with low MAO activity were distinguished by increased behavioral activity and reduced social apathy. Inpatients with low MAO activity were distinguished by severity of illness and symptoms. Hallucinations were significantly more frequent among patients with low MAO activity. The authors suggest that **platelet** MAO activity might decline in some actively schizophrenic patients as part of the psychotic process.

L24 ANSWER 150 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1978:199040 Document No.: BA66:11537. SUBSTRATE TYPIC CHANGES OF **PLATELET** MONO AMINE OXIDASE ACTIVITY IN SUBTYPES OF **SCHIZOPHRENIA**. DEMISCH L; VON DER MUEHLEN H; BOCHNIK H J; SEILER N. CENT. RECH. MERRELL INT., 16 RUE D'ANKARA, F-67084 STRASBOURG, FR.. ARCH PSYCHIATR NERVENKR, (1977 (RECD 1978)) 224 (4), 319-330. CODEN: APNVAV. ISSN: 0003-9373. Language: English.

AB Monoamine oxidase (MAO) activity was measured in the **platelets** of 42 controls and 49 schizophrenic patients of 3 subtypes, using .beta.-phenylethylamine, p-tyramine and tryptamine as substrates. Characteristic differences of MAO activity were observed between **platelets** of patients and controls; the differences were substrate-typic, i.e., decreased enzyme activity was found with all 3 substrates in **platelets** of the paranoid subtype. With tryptamine, MAO activity was decreased in the **platelets** of all 3 sub-types of **schizophrenia**. With p-tyramine, MAO activity was low in patients with affective psychoses and paranoid **schizophrenia**. The value of MAO activity measurements as a means for distinguishing sub-types of schizophrenic disorders was improved by using tryptamine and p-tyramine as substrates. Possible mechanisms of the substrate-typic changes of **platelet** MAO activity in **schizophrenia** were discussed.

L24 ANSWER 151 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

78112948 EMBASE Document No.: 1978112948. Pathology of folate deficiency. Hoffbrand A.V.. Roy. Free Hosp., London, United Kingdom. Proceedings of the Royal Society of Medicine 70/2 (82-84) 1977. CODEN: PRSMA4. Pub. Country: United Kingdom. Language: English.

AB Three main types of folate deficiency are distinguished: inadequate intake of the vitamin and malabsorption; treatment with a drug inhibiting folate metabolism, most commonly an inhibitor of the enzyme dihydrofolate reductase, e.g. methotrexate or pyrimethamine; and a lack of vit.B12. Although there is still discussion of the exact mechanism, it seems most probable that vit.B12 deficiency causes 'trapping' of folate in its plasma form, methyltetrahydrofolate. This is thought to occur because vit.B12 is needed as a cofactor in the homocysteine-methionine reaction by which a methyltetrahydrofolate entering cells from plasma is 'demethylated' to release tetrahydrofolate, from which all the intracellular folate coenzymes are made. A wide variety of tissues may be affected by all 3 types of folate deficiency (table), so that a number of clinical lesions

may occur. Folate is required in 3 reactions in DNA synthesis, one in pyrimidine synthesis (thymidylate synthesis) and 2 in purine synthesis. The organs most affected are the bone marrow, the epithelial cell surfaces and the gonads. The defects produced in the bone marrow include a fall in red cell, white cell and **platelet** counts, due partly to death of their precursors in the marrow (ineffective hemopoiesis) and partly to shortened survival of the cells that do reach the peripheral blood. The circulating red cells are macrocytic. These changes are identical whether folate or vit. B12 deficiency is the underlying cause. When the deficiency is acute and severe, as with large doses of methotrexate, thrombocytopenia and leukopenia are more likely to occur than anemia, because of the shorter life-span of neutrophils and **platelets** than red cells in the circulation. The bone marrow precursors, transformed lymphocytes and dividing epithelial cells all show chromosome abnormalities. Selective nutrient deficiency affecting one cell line rather than another, e.g. lymphocytes and not bone marrow, has been described. The epithelia may show glossitis, angular cheilosis and in some cases a malabsorption syndrome. Histologically, there are changes in the mouth epithelium and in the bronchi, bladder and cervix uteri. Again, changes localized to one tissue have been described. The jejunal mucosa is also affected, but probably not in pure nutritional folate deficiency. Acute folate deficiency (methotrexate) may cause ulceration of mouth and upper intestinal mucosa, but whether folate or B12 deficiency cause ulceration of the buccal or gut mucosa is uncertain. Severe folate deficiency causes sterility in both sexes. Methotrexate is indeed a powerful abortifacient. Less well established effects of folate deficiency in pregnancy are an increased frequency of congenital malformations. A poorly understood effect of folate deficiency is a reversible pigmentation of the skin. Regeneration of a cirrhotic liver is impaired in folate-deficient alcoholics and fibrosis of the liver occurs in patients with psoriasis given prolonged courses of methotrexate. Vit.B12 deficiency causes a fall in serum alkaline phosphatase to subnormal levels. Neurologic syndromes described include: mental retardation in inborn errors of folate metabolism, a peripheral neuropathy or posterior lateral column lesion in nutritional folate deficiency in the elderly, a **schizophrenia** -like illness in a child with homocystinuria and an inborn error of folate metabolism, and an impaired psychologic state with relative protection from fits in epileptics with folate deficiency. The extensive literature on all these aspects has recently been comprehensively reviewed by Reynolds (Clinics in Haematology, 1976). The best substantiated of these effects is the neurologic damage caused by methotrexate. Whether nutritional folate deficiency, even when severe, causes more than minor psychologic changes remains an open question.

L24 ANSWER 152 OF 160 MEDLINE

77071241 Document Number: 77071241. PubMed ID: 1036740. **Platelet** uptake and efflux of serotonin in subtypes of psychotic children. Rimland B. JOURNAL OF AUTISM AND CHILDHOOD SCHIZOPHRENIA, (1976 Dec) 6 (4) 379-82. Journal code: 1264240. ISSN: 0021-9185. Pub. country: United States. Language: English.

AB The only finding of a metabolic defect in psychotic children which has been replicated in a blind study is the discovery of an elevated efflux of serotonin from the **platelets** of children with early infantile autism (Boullin, Coleman, & O'Brien, 1970; Boullin, Coleman, O'Brien, & Rimland, 1971). The reported failure of Yuwiler, Ritvo, Geller, Glousman, Schneiderman, and Matsuno (1975) to replicate the Boullin et al. findings is attributable to differences in the method of selecting subjects. The Boullin et al. studies found that only children with classical infantile autism, as diagnosed by the Rimland E-2 check list, manifested the metabolic error. Since only 10% of psychotic children score in the autistic range on the check list, and since all children in the Yuwiler et al. study displayed the syndrome of "perceptual inconstancy," a syndrome

inconsistent with "insistence on the preservation of sameness," an integral part of the syndrome of early infantile autism as scored on the E-2 check list, the failure of Yuwiler et al. to find elevated efflux in their sample was to be expected.

L24 ANSWER 153 OF 160 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1976:233209 Document No.: BA62:63209. CEREBRO SPINAL FLUID ACID MONO AMINE METABOLITES AS A POSSIBLE REFLECTION OF CENTRAL MONO AMINE OXIDASE ACTIVITY IN CHRONIC **SCHIZOPHRENIA**. BOWERS M B JR. BIOL PSYCHIATRY, (1976) 11 (2), 245-249. CODEN: BIPCBF. ISSN: 0006-3223. Language: Unavailable.

AB Patients with a criterion **diagnosis** of **schizophrenia** or manic-depressive (bipolar) illness were studied. The schizophrenics were subdivided into acute and chronic subgroups with the distinction being made on the basis of an illness of > 12 mo. duration. Bipolar patients were grouped according to whether hospitalization was for treatment of mania or depression. Following a 3 wk period without medication, patients were given probenecid (100 mg/kg) in 6 divided doses over an 18 h period. Lumbar puncture was performed 24 h after the 1st dose; CSF was removed for analysis of 5-hydroxyindole acetic acid (5-HIAA) and homovanillic acid (HVA). Metabolite values for the various patient groups were compared using an unpaired t-test. There was lack of correlation between metabolites and probenecid; there seemed to be a near maximum inhibition of the transport system. Group differences were probably the result of differences in rates of metabolite formation. CSF HVA was significantly higher in bipolar manics compared to bipolar depressives ($P < 0.01$) and chronic schizophrenics ($P < 0.05$). CSF HVA in acute schizophrenics was higher than in chronic schizophrenics, 5 HIAA was elevated compared to bipolar depressives or acute schizophrenics and almost reached significance. A relatively high mean 5 HIAA and a relatively low mean HVA was found in groups of poor-prognosis schizophrenic patients. The patterns of CSF 5 HIAA and HVA in chronic **schizophrenia** are consistent with the hypothesis that a central Type B MAO [monoamine oxidase] deficiency may be associated, although relatively low values for both metabolites were also found in bipolar depression. Results are consistent with those of Meltzer et al. (1974) that MAO isoenzyme from **platelets** of schizophrenics might be a different species of Type B MAO.

L24 ANSWER 154 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
78004100 EMBASE Document No.: 1978004100. Blood **platelet** monoamine oxidase activity in schizophrenic children and their families. A preliminary study. Campbell M.; Friedman E.; Green W.H.; et al.. Dept. Psychiat., New York Univ. Med. Cent., New York, N.Y., United States. Neuropsychobiology 2/4 (239-246) 1976.
CODEN: NPBYAL. Language: English.

AB In this study monoamine oxidase (MAO) activity was measured in blood **platelets** of 21 individuals (age 2.5-19 years) who were diagnosed at preschool age as schizophrenics; MAO activity was not significantly different from that found in normals. An insignificant correlation was found between MAO activity in patients and age; a similar correlation for normals was also insignificant. In a sample of 15 families, no significant correlation between MAO activity of patients and their parents was obtained.

L24 ANSWER 155 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
78008894 EMBASE Document No.: 1978008894. Fluctuation of fluorescent material in the rat's and human leucocytes under various situations. Mato M.; Uchiyama Y.; Ookawara S.; et al.. Dept. Anat., Jichi Med. Sch., Minamikawachi, Tochigi, Japan. Acta Histochemica 57/2 (191-197) 1976.
CODEN: AHISA. Language: English.

AB When modified Falck-Hillarp technique was applied to blood smears of rat

and human specimens, the fluorescence was detected not only in **platelets**, but in leukocytes. In rat leukocytes and **platelets**, the bimodal daily rhythm of fluorescent material was observed under physiological conditions. The rhythm was modified considerably if animals were exposed to continuous lighting. The intensity and number of fluorescent cells in human smears increased markedly in patients suffering from **schizophrenia** compared with controls. The content of fluorescent material in leukocytes is assumed to be closely related with an activity of the living states.

L24 ANSWER 156 OF 160 MEDLINE DUPLICATE 37
76119277 Document Number: 76119277. PubMed ID: 2524. Reduction of blood **platelet** monoamine oxidase activity in schizophrenic patients on phenothiazines. Takahashi S; Yamane H; Tani N. FOLIA PSYCHIATRICA ET NEUROLOGICA JAPONICA, (1975) 29 (3) 207-14. Journal code: 0372774. ISSN: 0015-5721. Pub. country: Japan. Language: English.
AB A newly developed assay for monoamine oxidase (MAO) activity in blood **platelets** (serotonin used as substrate) was applied for the measurement of the enzyme activity in 76 schizophrenic patients. No significant reduction was found in the blood **platelet** MAO activity in a group of 33 untreated schizophrenic patients, as compared to that in the normal controls. Male patients revealed to have lower enzyme activity than females in the schizophrenic group, as we described previously in the normal subjects. Treatment with phenothiazines caused significant reduction of blood **platelet** MAO activity, while **platelet** serotonin content and **platelet** count appeared to be not affected by the drug treatment. The authors suggest that blood **platelet** MAO activity may be related to hormonal factors but not to psychiatric **diagnosis** of **schizophrenia** or constitution liable to schizophrenic illnesses.

L24 ANSWER 157 OF 160 MEDLINE
76153487 Document Number: 76153487. PubMed ID: 769113. Average evoked response augmenting/reducing in **schizophrenia** and affective disorders. Buchsbaum M. RESEARCH PUBLICATIONS - ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE, (1975) 54 129-42. Ref: 73. Journal code: 7505942. ISSN: 0091-7443. Pub. country: United States. Language: English.

L24 ANSWER 158 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 38
75172826 EMBASE Document No.: 1975172826. Blood **platelet** monoamine oxidase activity in psychiatric patients. Friedman E.; Shopsin B.; Sathananthan G.; Gershon S.. Neuropsychopharmacol. Res. Unit, Dept. Psychiat., New York Univ. Med. Cent., New York, N.Y. 10016, United States. American Journal of Psychiatry 131/12 (1392-1394) 1974. CODEN: AJPSAO. Language: English.
AB The authors measured monoamine oxidase (MAO) activity in blood **platelets** of normal control subjects, patients with **schizophrenia**, and patients with other psychiatric **diagnoses**. No differences were found among the group as a whole. However, in the control group, women had significantly higher enzyme activity than men. Female schizophrenic patients showed a trend toward lower activity than did female control subjects, but did not differ from the male schizophrenic patients. The authors suggest that **platelet** MAO activity may be related to hormonal, dietary, or genetic factors but not to psychiatric **diagnosis**.

L24 ANSWER 159 OF 160 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
76129437 EMBASE Document No.: 1976129437. Monoamine oxidase activity: a genetic marker of **schizophrenia**?. Owen F.; Ridges A.P.; Cookson I.B.. Dept. Psychiat., Univ. Liverpool, United Kingdom. Acta Geneticae Medicae et Gemellologiae Vol. 23/- (371-376) 1974. CODEN: AGMGAK. Language: English.

AB The results of pilot studies of the activities of **platelet** monoamine oxidase (MAO) and catechol O methyl transferase (COMT) in the blood of selected schizophrenics and the families of schizophrenics is presented. No statistically significant difference was found between the blood COMT levels of 21 control subjects and 26 schizophrenics, whereas the values found for **platelet** MAO activity were significantly lower for the schizophrenic group than for the control group. In one acutely disturbed first admission schizophrenic the **platelet** MAO activity increased to a normal level in parallel with the clinical improvement, whereas in the relapsing schizophrenics the **platelet** MAO activity remained at its initial level although the clinical picture improved. No consistent findings with regard to the **platelet** MAO activity emerged from the study of 3 families having a history of **schizophrenia**.

L24 ANSWER 160 OF 160 MEDLINE
73253289 Document Number: 73253289. PubMed ID: 4581281. Serotonin and central nervous system syndromes of childhood: a review. Coleman M. JOURNAL OF AUTISM AND CHILDHOOD SCHIZOPHRENIA, (1973 Jan-Mar) 3 (1) 27-35. Ref: 29. Journal code: 1264240. ISSN: 0021-9185. Pub. country: United States. Language: English.

=> s (shinitzky m?/au or deckmann m?/au)
L25 766 (SHINITZKY M?/AU OR DECKMANN M?/AU)

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L26 40 L25 AND SCHIZOPHRENIA

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L27 20 DUP REMOVE L26 (20 DUPLICATES REMOVED)

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L27 ANSWER 1 OF 20 CAPLUS COPYRIGHT 2003 ACS
2002:736274 Document No. 137:259655 Novel peptides for the diagnosis of **schizophrenia**. **Deckmann, Michael** (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 2002074793 A2 20020926, 27 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-IL233 20020321. PRIORITY: IL 2001-142159 20010321; US 2001-PV278659 20010321.

AB Short peptides are provided, which bind to a body fluid sample obtained from a schizophrenic patient at a substantively higher level than to a body fluid sample obtained from a non-schizophrenic individual. The peptides are no more than 10 amino acids long and comprise a continuous sequence of at least 5 amino acids which consists of at least one pos. charged amino acid at one of its ends. The provided peptides, which are the putative binding sites of autoantibodies found in high levels in schizophrenic individuals, are thus useful in diagnosis of **schizophrenia**. Biotin-labeled peptide LVVGLCK was coated onto streptavidin-coated tubes and used to test plasma samples of schizophrenic patients and control non-schizophrenic patients in an enzyme immunoassay.

L27 ANSWER 2 OF 20 MEDLINE DUPLICATE 1

2002359435 Document Number: 22097683. PubMed ID: 12104086. A conformational epitope which detects autoantibodies from schizophrenic patients. **Deckmann Michael**; Mamillapalli Ramanaiah; Schechtman Ludmila; **Shinitzky Meir**. (Neurogenic Ltd., P.O. Box 29866, 61298 Tel Aviv, Israel.) CLINICA CHIMICA ACTA, (2002 Aug) 322 (1-2) 91-8. Journal code: 1302422. ISSN: 0009-8981. Pub. country: Netherlands. Language: English.

AB We previously found autoantibodies against platelets in schizophrenic patients. One of the platelet proteins that bind these antibodies is enolase. Here, we describe the isolation and sequencing of an immunoreactive peptide after enzymatic digestion of enolase. The 3-D structure of enolase indicates that, unexpectedly, this peptide is buried inside the protein. However, 3-D surface analysis leads to the identification of a conformational epitope that resembles the binding peptide and might constitute a specific binder of the autoantibodies. In a screening of antibody binding with the peptide LVVGLCK, we found in 50 serum samples of controls a mean of O.D.=0.46; s= +/- 0.21 relative enzyme immunoassay units, while in sera of 39 schizophrenic patients, we found a mean of O.D.=1.47; s= +/- 0.65; P<0.0001. Furthermore, an inverse correlation was observed between duration of **schizophrenia** and the level of the detected autoantibodies. A screening of autoantibodies in sera of various mental disorders with this peptide is currently in progress.

L27 ANSWER 3 OF 20 CAPLUS COPYRIGHT 2003 ACS

2000:706969 Document No. 133:261536 Pharmaceutical compositions comprising cyclic glycerophosphates and analogs thereof for promoting neural cell differentiation. **Shinitzky, Meir** (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 2000057865 A2 20001005, 42 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-IL185 20000324. PRIORITY: IL 1999-129178 19990325.

AB Cyclic glycerophosphates and analogs thereof (CGs) are shown to exert neural promoting activities in target cells. Such activities include promotion of neuronal outgrowth, promotion of nerve growth, provision of dopaminotrophic supporting environment in a diseased portion of the brain, prevention of nerve degeneration and nerve rescue. These activities of the CGs render them useful for treatment of various disorders including but not limited to mental disorders such as, for example, **schizophrenia**, dementia or disorders resulting in learning disabilities. In addn., these CGs may be used for the treatment of neurodegenerative conditions such as Alzheimer's disease, Parkinson's disease, conditions resulting from exposure to harmful environmental factors or resulting from a mech. injury. The CGs may also be used to treat an individual suffering from a primary neurodegenerative condition in order to prevent or reduce the appearance of secondary degeneration in addnl. nerves ("nerve rescue"). For example, neural outgrowth of PC12 cells was seen when cells were grown in the presence of nerve growth factor (50 ng/mL) or 1,3-cyclic glycerophosphate (1 .mu.M), but not in the presence of linear .alpha.-glycerophosphate.

L27 ANSWER 4 OF 20 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

2000:299632 Document No.: PREV200000299632. Diagnosis of the susceptibility of contracting **schizophrenia**. **Shinitzky, Meir (1)**. (1) Kfar Shmaryahu Israel. ASSIGNEE: Yeda Research and Development Co. Ltd.,

Rehovot, Israel. Patent Info.: US 6008001 December 28, 1999. Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 28, 1999) Vol. 1229, No. 4, pp. No pagination. e-file. ISSN: 0098-1133. Language: English.

AB There is described an assay for the diagnosis of a mental disorder in an individual. A blood sample, a platelet-containing fraction thereof, or a fraction containing platelet-associated antibodies (PAA) shed from the platelets is withdrawn from the individual to be diagnosed. The withdrawn sample is contacted with an anti-human immunoglobulin antibody lacking the Fc domain (Fc-less anti-hIg antibody) and the degree of binding thereof to the PAA is determined. A degree of binding above that found in normal individuals indicates that diagnosed individual has a high likelihood of having a mental disorder.

L27 ANSWER 5 OF 20 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1999:392958 Document No.: PREV199900392958. Use of immunosuppressive agents for the treatment of **schizophrenia**. **Shinitzky, Meir** (1) ; **Deckmann, Michael**. (1) Kfar Shmaryahu Israel. ASSIGNEE: Yeda Research and Development Co., Ltd.. Patent Info.: US 5912250 Jun. 15, 1999. Official Gazette of the United States Patent and Trademark Office Patents, (Jun.15, 1999) Vol. 1223, No. 3, pp. NO PAGINATION. ISSN: 0098-1133. Language: English.

L27 ANSWER 6 OF 20 CAPLUS COPYRIGHT 2003 ACS

1999:659490 Document No. 131:270940 Assay for the diagnosis of **schizophrenia** based on a new peptide. **Shinitzky, Meir; Deckmann, Michael** (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9951725 A2 19991014, 37 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-IL190 19990330. PRIORITY: IL 1998-123925 19980402.

AB The invention concerns peptides which bind antibodies that are found in elevated levels in body fluids of schizophrenic patients and are found at a lower level or not found at all in body fluids of non-schizophrenic individuals. Using a computerized program, the antigenic epitope of the peptides of the invention is predicted as having a core of hydrophobic amino acids which is surrounded by pos. charged amino acids. The peptides of the invention are useful in the diagnosis of **schizophrenia** in an individual.

L27 ANSWER 7 OF 20 CAPLUS COPYRIGHT 2003 ACS

1999:390463 Document No. 131:16115 Skin test for **schizophrenia**. **Shinitzky, Meir; Deckmann, Michael** (Yeda Research and Development Co. Ltd., Israel). PCT Int. Appl. WO 9930163 A1 19990617, 21 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1998-IL592 19981207. PRIORITY: IL 1997-122490 19971207.

AB A diagnostic method for assaying **schizophrenia** in a subject is provided wherein a prepn. comprising platelet derived proteins or fractions thereof having a pI above about 6.5 is injected into a subject and the occurrence of delayed type hypersensitivity (DTH) reaction at the

site of the injection is detd. A pos. DTH reaction indicates that the tested subject has a high likelihood of being schizophrenic. The protein prepn. used in the diagnostic method is also provided as well as a method for its prepn. and a kit for use in the diagnosis of **schizophrenia** using the above method.

L27 ANSWER 8 OF 20 MEDLINE DUPLICATE 2

2000059338 Document Number: 20059338. PubMed ID: 10591989. Elevated cellular immune response to human heat-shock protein-60 in schizophrenic patients. Leykin I; Spivak B; Weizman A; Cohen I R; **Shinitzky M.** (Department of Biological Chemistry, Weizmann Institute of Science, Rehovot, 76100, Israel.) EUROPEAN ARCHIVES OF PSYCHIATRY AND CLINICAL NEUROSCIENCE, (1999) 249 (5) 238-46. Journal code: 9103030. ISSN: 0940-1334. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Heat shock protein-60 (HSP60) is implicated in several autoimmune diseases as a triggering antigen. Based on the autoimmune hypothesis of **schizophrenia,** we examined cellular and humoral responses against HSP60 and a series of its peptide fragments with peripheral blood samples of schizophrenic patients and healthy subjects each of group size between 12 to 32 participants. The average stimulation indices of peripheral blood mononuclear cells (PBMC) to HSP60 were 3.17 +/- 0.36 (mean +/- SE) for schizophrenic patients and 2.23 +/- 0.24 (mean +/- SE) for healthy subjects, with a significant difference between the groups (P = 0.0457). In parallel, 38 synthetic peptide fragments of HSP60, each of 18-21 amino acids, were tested for in vitro sensitization of PBMC. With one peptide (p32) the average stimulation index of PBMC from schizophrenic patients was significantly higher than that obtained for PBMC of control subjects (P = 0.0006). Comparing the cellular immune response to p32 between patients who were distinctive responders (n = 10) or non-responders (n = 10) to neuroleptic treatment indicated a similar elevation of cellular response in these groups. Antibodies against HSP60 were screened by dot-blot and ELISA in the sera of the above blood samples. Titers of IgG and IgM against HSP60 were found to be of similar magnitude in schizophrenic patients and in controls. Titers of IgA against HSP60 were somewhat higher in the sera of schizophrenic patients in comparison to sera of control subjects (P = 0.0605).

L27 ANSWER 9 OF 20 MEDLINE DUPLICATE 3

1998057797 Document Number: 98057797. PubMed ID: 9396015. Side effect profile of azathioprine in the treatment of chronic schizophrenic patients. Levine J; Gutman J; Feraro R; Levy P; Kimhi R; Leykin I; **Deckmann M;** Handzel Z T; **Shinitzky M.** (Beer Sheva Mental Health Center, Faculty of Health Sciences, Ben Gurion University of the Negev, Beer Sheva.) NEUROPSYCHOBIOLOGY, (1997) 36 (4) 172-6. Journal code: 7512895. ISSN: 0302-282X. Pub. country: Switzerland. Language: English.

AB Various findings suggest auto-immune changes in **schizophrenia.** We have recently demonstrated that platelets from schizophrenic patients bear autoantibodies (PAA) which cross-react with brain antigens. Accordingly, treatment of **schizophrenia** with an immunosuppressant might be of potential benefit. In a recent case study, a chronic schizophrenic patient treated with azathioprine has demonstrated a clear psychiatric improvement preceded by a decrease in PAA level. A phase I study designed for assessing side-effects of short-term azathioprine treatment in a group of schizophrenic patients is described here. From a group of 40 chronic non-responsive patients, 14 patients demonstrating high PAA level have entered the study and 11 have complied all along. Two groups were tested in parallel. In the first (6 patients) 150 mg/day was given for 7 weeks while in the second (5 patients) the same regimen was given for two periods of 7 weeks with an interval of 6 weeks. Blood biochemistry and cell count, as well as determination of PAA were

carried out weekly, starting 3 weeks before the trial and continuing up to 7 weeks after the treatment. Two out of 11 patients developed leucopenia in week 4. No other side-effects were recorded in any of the patients. A substantial reduction in PAA was observed in 3 out of 6 patients in group I and 4 out of 5 in group II. Two patients showed improvement of psychiatric symptomatology. Our results demonstrate that short-term azathioprine treatment induces transient leucopenia in 18% of the patients receiving the drug, much alike the percentage reported for other patient populations.

L27 ANSWER 10 OF 20 MEDLINE DUPLICATE 4
97431153 Document Number: 97431153. PubMed ID: 9285246. Short and long-term immunosuppressive effects of clozapine and haloperidol. Leykin I; Mayer R; **Shinitzky M.** (Department of Membrane Research and Biophysics, Weizmann Institute of Science, Rehovot, Israel.) IMMUNOPHARMACOLOGY, (1997 Aug) 37 (1) 75-86. Journal code: 7902474. ISSN: 0162-3109. Pub. country: Netherlands. Language: English.

AB In line with the autoimmune hypothesis of **schizophrenia** we have tested in this study whether the commonly used neuroleptics, clozapine and haloperidol can also act as systemic immunosuppressants. Twenty one hospitalized chronic schizophrenic patients participated in the study. Five were free of neuroleptic treatment while the other 16 were under chronic treatment with either clozapine (n = 8), or haloperidol (n = 8). Fourteen age matched normal subjects served as the control group. Conventional in vitro mitogenic stimulation of peripheral blood lymphocytes with phytohaemagglutinin (PHA) indicated a clear suppression of responsiveness of approximately 50% in all treated patients. The PHA response of the untreated patients was virtually identical to that of the control group. The in vitro effect of haloperidol and clozapine on PHA stimulation of lymphocytes from normal subjects was determined by 3H-thymidine uptake and secretion of interleukin-2, interleukin-4 and interferon-gamma. Both clozapine and haloperidol suppressed thymidine incorporation and cytokine secretion at a drug concentration of above 1 microM, reaching full suppression at 50 microM. Similar suppressive effects of clozapine and haloperidol were also observed in mixed lymphocyte reaction of mouse lymphocytes. Assays with radioactive ligands indicated that clozapine is not incorporated into the lymphocytes but presumably exerts its action by binding to specific surface sites. The long term immune suppression induced by neuroleptic treatment may inhibit putative autoimmune responses against neurological sites and could thus act synergistically with the direct antagonistic action on brain receptors for the overt amelioration of psychotic behaviour.

L27 ANSWER 11 OF 20 CAPLUS COPYRIGHT 2003 ACS
1996:123755 Document No. 124:156032 Use of immunosuppressive agents for the treatment of **schizophrenia**. **Shinitzky, Meir; Deckmann, Michael** (Yeda Research and Development Co., Ltd., Israel). PCT Int. Appl. WO 9534306 A1 19951221, 23 pp. DESIGNATED STATES: W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1995-EP2289 19950613. PRIORITY: IL 1994-110011 19940613.

AB The invention relates to a pharmaceutical compn. for the treatment of schizophrenic disorders which comprises a pharmaceutically acceptable carrier and as active ingredient an immunosuppressive agent.

L27 ANSWER 12 OF 20 CAPLUS COPYRIGHT 2003 ACS
1995:934131 Document No. 123:337435 Assay for the diagnosis of **schizophrenia**. **Shinitzky, Meir; Deckmann, Michael** (Yeda Research and Development Co., Ltd., Israel; Rycus,

Avigail). PCT Int. Appl. WO 9523970 A1 19950908, 19 pp. DESIGNATED STATES: W: AU, BR, CA, JP, US; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1995-US2426 19950228. PRIORITY: IL 1994-108789 19940301; IL 1994-110142 19940628.

AB An immunol. assay for the diagnosis of **schizophrenia** in an individual is described. The assay comprises the following steps: (a) a blood sample, a platelet-contg. fraction of a blood sample, or a fraction contg. platelet-assocd. antibodies (PAA) shed from the platelets is obtained from an individual; (b) the sample is contacted with platelet antigens fixed to a solid support, and subsequently with an antibody detection system; and (c) the binding pattern of the PAA to the platelet antigens is detd. and compared to the binding pattern of a sample obtained from a normal individual. A difference in patterns indicates that the individual has a high likelihood of having **schizophrenia**. The assay is capable of differentiating **schizophrenia** from dementia, as well as from Idiopathic Thrombocytopenia Purpura (ITP), an autoimmune disease directed against a platelet antigen.

L27 ANSWER 13 OF 20 MEDLINE DUPLICATE 5

96127463 Document Number: 96127463. PubMed ID: 8563786. Number of platelet dense granules varies with age, **schizophrenia** and dementia. Kessler A; **Shinitzky M**; Kessler B. (Department of Membrane Research, Weizman Institute of Science, Rehovot, Israel.) DEMENTIA, (1995 Nov-Dec) 6 (6) 330-3. Journal code: 9010348. ISSN: 1013-7424. Pub. country: Switzerland. Language: English.

AB In the present study we observed that the number of dense granules per platelet increases with age, attaining a maximum level above the age of about 40 years. Platelets of newborns apparently contain only a small number of dense granules per platelet. The numbers of platelet dense granules and platelet cell size in schizophrenic patients increase compared to age-matched healthy controls. In contrast, in Alzheimer-type dementia the number of platelet dense granules tends to decrease compared to healthy persons.

L27 ANSWER 14 OF 20 MEDLINE

94285617 Document Number: 94285617. PubMed ID: 7912324. Treatment of **schizophrenia** with an immunosuppressant. Levine J; Susnovski M; Handzel Z T; Leykin I; **Shinitzky M**. LANCET, (1994 Jul 2) 344 (8914) 59-60. Journal code: 2985213R. ISSN: 0140-6736. Pub. country: ENGLAND: United Kingdom. Language: English.

L27 ANSWER 15 OF 20 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 6

94208481 EMBASE Document No.: 1994208481. Treatment of **schizophrenia** with an immunosuppressant [7]. Levine J.; Susnovski M.; Handzel Z.; Leykin I.; **Shinitzky M.**. Abarbanel Mental Health Centre,Bat-Yam, Israel. Lancet 344/8914 (59-60) 1994.
ISSN: 0140-6736. CODEN: LANCAO. Pub. Country: United Kingdom. Language: English.

L27 ANSWER 16 OF 20 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

94014370 EMBASE Document No.: 1994014370. Platelets from schizophrenic patients bear autoimmune antibodies that inhibit dopamine uptake. Kessler A.; **Shinitzky M.**. Membrane Research/Biophysics Dept., Weizmann Institute of Science,76100 Rehovot, Israel. Psychobiology 21/4 (299-306) 1993.
ISSN: 0889-6313. CODEN: PSYBEC. Pub. Country: United States. Language: English. Summary Language: English.

L27 ANSWER 17 OF 20 MEDLINE DUPLICATE 7

91315044 Document Number: 91315044. PubMed ID: 1859087. Platelet autoantibodies in dementia and **schizophrenia**. Possible

implication for mental disorders. **Shinitzky M; Deckmann M;** Kessler A; Sirota P; Rabbs A; Elizur A. (Department of Membrane Research, Weizmann Institute of Science, Rehovot, Israel.) ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1991) 621 205-17. Journal code: 7506858. ISSN: 0077-8923. Pub. country: United States. Language: English.

AB Platelets isolated from blood of demented and schizophrenic patients were found to bear surface antibodies at a considerably higher titer than those found on platelets from normal age-matched groups or patients with affective disorders. The platelet count in demented and schizophrenic patients correlated inversely with the level of the platelet associated antibodies (PAA) which suggested an autoimmune route of opsonization. In most individual cases of dementia or **schizophrenia** PAA and platelet count were found to oscillate with time between high PAA-low platelet number and low PAA-high platelet number in approximately inverse correlation. PAA isolated from demented patients were found to cross-react with platelets from normals and with brain tissue from rats. Furthermore, molecular weights of specific brain antigens were identified by binding to PAA. These observations support the possibility that PAA might be implicated in the etiology of some mental dysfunctions associated with dementia and **schizophrenia**.

L27 ANSWER 18 OF 20 SCISEARCH COPYRIGHT 2003 ISI (R)

91:480131 The Genuine Article (R) Number: FZ453. PLATELET AUTOANTIBODIES IN DEMENTIA AND **SCHIZOPHRENIA** - POSSIBLE IMPLICATION FOR MENTAL-DISORDERS. **SHINITZKY M (Reprint); DECKMANN M;** KESSLER A; SIROTA P; RABBS A; ELIZUR A. WEIZMANN INST SCI, DEPT MEMBRANE RES, IL-76100 REHOVOT, ISRAEL (Reprint); ABARBANEL MENTAL HOSP, BAT YAM, ISRAEL. ANNALS OF THE NEW YORK ACADEMY OF SCIENCES (1991) Vol. 621, No. JUL, pp. 205-217. Pub. country: ISRAEL. Language: ENGLISH.

L27 ANSWER 19 OF 20 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1992:160375 Document No.: BR42:76575. AUTOIMMUNE **SCHIZOPHRENIA** NOVEL FACTS ABOUT AN OLD HYPOTHESIS. KESSLER A; **SHINITZKY M.** DEP. MEMBRANE RES. AND BIOPHYSICS, THE WEIZMANN INST. OF SCIENCE, ROHOVOT.. THIRD INTERNATIONAL CONGRESS ON NEUROIMMUNOLOGY, JERUSALEM, ISRAEL, OCTOBER 27-NOVEMBER 1, 1991. J NEUROIMMUNOL. (1991) 0 (SUPPL 1), 81. CODEN: JNRIDW. ISSN: 0165-5728. Language: English.

L27 ANSWER 20 OF 20 CAPLUS COPYRIGHT 2003 ACS

1987:561689 Document No. 107:161689 A lipid-phospholipid mixture for membrane fluidization and its use. **Shinitzky, Meir** (Yeda Research and Development Co., Ltd., Israel). Eur. Pat. Appl. EP 213724 A1 19870311, 16 pp. DESIGNATED STATES: R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE. (English). CODEN: EPXXDW. APPLICATION: EP 1986-305736 19860725. PRIORITY: US 1985-759270 19850726.

AB Membrane fluidity is increased by administration of a 7:3 mixt. of neutral lipids-phospholipids. The neutral lipids are glycerides, esp. triglycerides. The phospholipids consist of phosphatidyl choline (I) and phosphatidyl ethanolamine (II), preferably in a 2:1 ratio. This compn. is useful for treating a wide variety of disorders which are mediated by membrane lipid imbalance. Neutral lipids extd. from egg yolks were mixed with I and II in a 7:2:1 ratio. This mixt. increased the fluidity of human erythrocytes and lymphocytes, and extd. cholesterol from human lymphocytes, in vivo. Non-diseased immune-suppressed humans >75 yr old were treated with this mixt. orally for several weeks. During the test period, the responsiveness of peripheral blood lymphocytes to mitogens increased to a level typical of that found in the young. Upon cessation of this supplement, the lymphocyte responsiveness declined towards the initial level.

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